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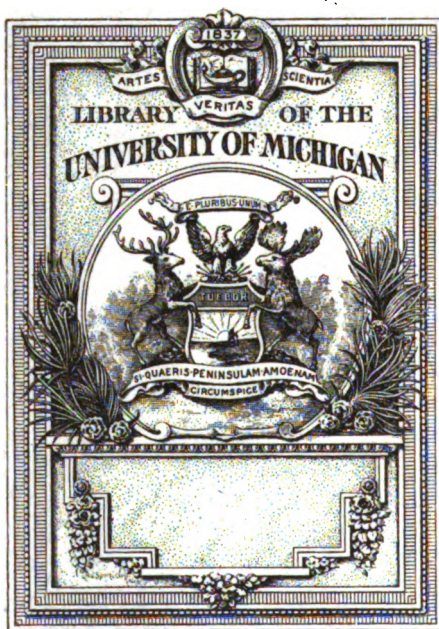
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# INTERNATIONAL CLINICS:

A QUARTERLY OF CLINICAL LECTURES

ON

MEDICINE, NEUROLOGY, SURGERY, GYNÆCOLOGY,  
OBSTETRICS, OPHTHALMOLOGY,  
LARYNGOLOGY, PHARYNGOLOGY, RHINOLOGY,  
OTOLOGY, AND DERMATOLOGY,

*AND SPECIALLY PREPARED ARTICLES ON TREATMENT AND DRUGS.*

BY PROFESSORS AND LECTURERS IN THE LEADING  
MEDICAL COLLEGES OF THE UNITED STATES,  
GERMANY, AUSTRIA, FRANCE, GREAT  
BRITAIN, AND CANADA.

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VOLUME II. EIGHTH SERIES. 1898.

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# Drugs and Remedial Agents.

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## SUGGESTIONS AS TO THE USE OF DIGITALIS.

BY J. N. HALL, M.D.,

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I know of no important drug concerning which so many misapprehensions exist in the minds of the members of the profession as may be found in the case of digitalis. I wish to indicate some of the conditions under which it should be used, and some of those under which its use is distinctly contraindicated.

Let us briefly review the most important physiologic and toxic effects of the drug upon the circulation. All authorities agree that it lessens the frequency of the heart-beat considerably in physiologic doses, and greatly in toxic ones. This lessening in the number of beats per minute is brought about solely by the prolongation of the diastole. With the physiologic dose the amount of work done by the heart is increased, but as we approach the toxic stage it is decreased, owing, apparently, to the imperfect diastole, and consequent non-admission of blood to the cavities of the heart. During the time of physiologic action the blood-pressure is raised, decreasing as poisoning supervenes.

Coincident with the increase in blood-pressure is the contraction of the peripheral arterioles, due in part to a direct action upon their walls, and in part to its action upon the vasomotor centre in the medulla. The diuretic action of digitalis is due to the increased blood-pressure in the glomeruli of the kidney, and is more marked, naturally, in conditions of low arterial pressure.

Most of the benefits conferred by digitalis arise from the increase of blood-pressure to which it gives rise. Yet many authors object to the drug because it contracts the arterioles. We have noted that a moderate increase in the amount of work done by the



heart results from its administration. It seems to have been overlooked by certain writers that it would be impossible to raise the general blood-pressure materially without hindering, in some manner, the passage of blood through the arterioles. Traube, quoted by Wood, "found that if the spine be divided, digitalis is powerless to increase the blood-pressure, though lessening as usual the pulse-rate." The separation of the vasomotor nerve centre from the small vessels is evidently responsible for this action.

It is well known that amyl nitrite acts as a powerful stimulant to the action of the heart, but dilating the peripheral arterioles widely, as it does, its final action is to cause a rapid fall in blood-pressure. If, however, the aorta be tied, the blood-pressure above the ligature rises greatly. By analogy, we may readily see that, without its action in contracting the arterioles, digitalis would lose its distinctive character as the best agent we have for raising the arterial pressure in those conditions in which, owing to some defect in the circulatory system, it is below normal. We may sum up this matter by saying that, with a moderate increase in power, we cannot raise the pressure in a system of pipes if the outlet remains perfectly free.

It is often proposed to administer some one of the nitrites, or the spirit of nitrous ether, with digitalis, to obviate the contraction of the arterioles. If the object be to overcome an abnormal contraction of these vessels, or to obtain the advantage of slowing the pulse without increasing the blood-pressure, as in the treatment of aneurism, this procedure is rational. If it be done, however, with the idea that the beneficial effects of raising a lowered blood-pressure are to be obtained, we must conclude that, if this pressure be actually raised, an insufficient dosage of the opposing drug has been used to produce any material dilatation of the vessels in question.

Much of the reasoning in regard to the use of digitalis in aortic regurgitation is similarly faulty, especially as to the effect of the lengthened diastole and the increased pressure upon the left ventricle. For example, the following paragraph has, by some curious error, been admitted to Butler's excellent "Text-Book of Materia Medica, Therapeutics, and Pharmacology:" "The regurgitant stream has probably little or no influence in producing the dilatation (of the left ventricle), since the cubic area of the ventricular cavity covered by the stream is so much greater than that of its inlet that

it is difficult to see how great pressure could be exerted in this way." This statement is in direct contravention of the principles upon which the hydraulic press is constructed, and is, therefore, utterly false. "Pressure exerted anywhere upon a mass of liquid is transmitted undiminished in all directions." (Ganot's "Physics.")

It is objected to the use of digitalis in aortic insufficiency that, by lengthening the period of diastole, it allows more time during which regurgitation may take place. It is not commonly noted that the auricle has likewise a lengthened interval of rest, during which it becomes thoroughly filled, and better able to fill in turn the left ventricle under an increased tension; nor that the yielding of the ventricular wall to the diastolic pressure must be less because of the very improvement in tone and power which has enabled it to raise the arterial tension. This operates to the advantage of the heart by permitting a smaller reflux of blood, to be moved at the next systole. It is certain that, in many cases, the benefit to the circulation in aortic regurgitation, as in many other conditions, results much more from the improvement in the nutrition of the myocardium, owing to its greater period of repose during the lengthened diastole, to the greater interval for the filling of the coronary arteries, and the increased tension under which they are filled, than from a mere compulsion, under the influence of the digitalis, to perform, temporarily, an increased amount of work. Some writers overlook this element in the case entirely. In my opinion, the theoretical objection to the use of digitalis in the disease under consideration should not for an instant prevent its administration in cases in which, were any other valve affected, we should consider it indicated.

We are repeatedly cautioned not to use digitalis in case of fatty degeneration of the cardiac muscle, for fear that increased tension may cause a rupture of the wall, or that asystole may result. If the heart-action be slow, it is better, no doubt, to use other cardiac tonics, rather than one which is known to still further slow the pulse. With a rapid heart-action, however, if the circulation really demands the use of a drug which shall raise the blood-tension, I have always felt that it was entirely proper to give digitalis a careful trial. The patient has some chance of improvement as regards the nutrition of his myocardium from the increase of tension, as already mentioned in the case of aortic regurgitation, and this possibility of thus improving his general circulation more than offsets, in my

opinion, the possible danger of rupture of the cardiac wall. If the patient really needs the digitalis, the danger mentioned is to be chosen rather than the certainty of death from gradual failure of the circulation.

Wood long ago pointed out the danger in the use of digitalis in aneurism, the increase in the intravascular tension more than offsetting the decrease in the number of pulsations to which the aneurismal sac is subjected. This author states that he has seen sudden death from rupture of the sac from the administration of the drug in question.

Renaud<sup>1</sup> has called attention to the fact that if a tachycardia is apparently permanent, and not influenced by the free administration of digitalis, it is probably to be accounted for by compression of the pneumogastric nerve by aneurism of the aorta, cancerous tumor, lymphadenoma or otherwise. He states "that the resistance of this tachycardia to digitalis constitutes one of the principal elements in the diagnosis."

We still find physicians occasionally prescribing digitalis for hæmoptysis, of pulmonary as distinguished from cardiac origin. This bleeding may come from the rupture of congested capillaries about a focus of disease, generally tuberculous, or from the opening of an artery by the spread of an ulcerative process to it during the formation of a cavity. The latter form is very commonly fatal, while the former variety generally ceases without doing serious harm. It is, indeed, a question whether it is not actually advantageous, through diminution of the congestion of the affected area, for Flint and other writers have shown that the percentage of recoveries is greater among those tuberculous patients who have suffered from hæmoptyses. I cannot conceive how digitalis can be of service in these cases, for its action in raising the vascular tension must more than offset the slowing of the pulse and the contraction of the smaller vessels. In this class of cases particularly, it is extremely difficult to estimate the precise action of the drug exhibited, for we all know that in many such cases the bleeding is arrested before the arrival of the physician, and the natural tendency is certainly towards cessation.

Germain Sée has advanced reasons for believing that digitalis raises the tension in the pulmonary artery to a greater extent, pro-

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<sup>1</sup> Troubles fonctionnels du Cœur, Paris, 1893.

portionally, than that in the systemic arteries. It would seem more wise to adopt the plan followed by nature in the giving of our remedies, seeking to lower vascular tension, as in the syncope following severe hemorrhages. Aconite may probably be given in these cases with more reason than digitalis, or even ergot, for the action of the latter drug is quite similar, in regard to the points under discussion, to that of digitalis. Until we know the exact cause of the hemorrhage, it must remain a very difficult question to decide what drug will act most beneficially. Meanwhile, we must remember that an agent which contracts the arterioles to one-half of their previous diameter, perhaps slows the pulse one-half, and yet raises the blood-pressure to double the normal height, can do little, if anything, towards checking bleeding. If we cannot obtain empirical support for the giving of any one drug, and this would be possible in this matter only by the collection of an enormous number of cases, we must follow out the theoretical indications.

To my mind, all the reasoning thus far advanced applies with yet greater force in cases of the graver form of hemorrhage from ulceration into an artery, because of the greater size of the vessel. I believe that digitalis is never indicated in this disease.

It is altogether different, however, when we come to hemorrhage from the lungs in disease of the mitral valve. We have to deal here with a passive hemorrhage, due to the obstruction to the passage of the blood out of the lung, because of a "backing-up" of the current from the heart. Digitalis is here distinctly indicated, not for the checking of the immediate hemorrhage, but because it will, by increasing the vascular tension and improving the condition of the whole circulatory apparatus, abolish, more or less completely, the stagnation existing in the pulmonic as in the systemic circulation. In this way it will at least assist in the avoidance of future hemorrhages. It should be noted that one reason for failure to act immediately in checking an hæmoptysis may be the extreme slowness of absorption and action of digitalis in comparison with many other drugs. It would be well to use the tincture subcutaneously in an emergency.

We are told that extensive atheroma is a contraindication to the use of digitalis. It is undoubtedly more dangerous to use the drug in such a condition of the arteries than under normal conditions, for the increase in tension may rupture a weakened cerebral vessel.

On the other hand, an abnormally low arterial tension is as dangerous to such a patient as to any other. In my opinion the patient had better assume the risks associated with measures for the raising of vascular tension to a point consistent with the continuance of his organic functions than to incur the certain dangers which will otherwise overtake him. Although calling for increased care in the administration of the drug, I believe that cases of marked atheroma should receive it whenever the circulatory condition demands an agent which shall increase vascular tension.

I wish to emphasize the value of this drug in pneumonia, long advocated, but not so widely used as it deserves to be. I have, clinically, I believe, seen much good result from its administration at the period, so commonly noted, when the right heart flags in its effort to force the blood through the obstructed lung. The possible selective action of digitalis for the right heart may here be of great advantage.

We hear so much discussion regarding the cumulative action of digitalis that I wish to enforce upon your attention one or two points which deserve consideration. I have noted a wide-spread impression among certain physicians that dangerous consequences may ensue if the dosage of the tincture is pushed beyond fifteen or twenty drops three times a day. My experience certainly corresponds with that of Wood, who has done so much in instructing us as to the use of digitalis,—that is, that we may give what seem like enormous doses where the emergency exists, not only with entire safety, but with great advantage to the patient. So long as the pulse is not dangerously slowed I believe we may push the drug fearlessly, provided the individual doses be not too large. It is well, however, to regard the usual cautions,—viz., to interrupt the administration for a few days occasionally to allow the system to clear itself; to give large doses only with care, during high fever, because of the danger of excessive action in case of a sudden fall of the temperature; to stop the use of the drug before tapping for ascites, for fear of sudden absorption of the active principles from the tissues after the operation; and to cease giving it in case of marked decrease in the amount of urine excreted, since its elimination is then greatly interfered with.

We occasionally see two cases of valvular disease apparently demanding precisely the same treatment, and give to each the same

preparation of digitalis in the same dose. One improves as expected, while the other complains of an increase in the oppression within the chest, and a failure as regards improvement in all the other symptoms. How are we to explain such discrepancy?

It is probably because digitalis sometimes, if not generally, acts more forcibly upon the ventricle than upon the auricle. Supposing the left auricle, in mitral regurgitation, to be more dilated than usual, and its muscular tissue in a more advanced stage of degeneration than that of the ventricle, we may see that the drug will act more efficiently upon the latter, and will thus increase the regurgitation and the intra-auricular pressure. The auricle meanwhile derives little benefit from the digitalis because of its degenerated myocardium, which is not likely to improve in tone under the treatment. As a result the auricle dilates still further, and its last condition is worse than its first. We are obliged to stop the administration of the digitalis. I have seen excellent results from the use of strychnine in some of these cases, if given in fairly large dose. In one case of aortic regurgitation, with probably secondary mitral insufficiency, digitalis increased the patient's discomfort decidedly. After the use of strychnine for some weeks, an attempt to use the former drug was again made, with the same result as at first. For nearly three years she has been doing fairly well under the almost constant use of strychnine, with occasional use of other tonics.

It is quite possible that some idiosyncrasy explains the intolerance of digitalis in some of these cases.

I agree entirely with those who recommend the use of the leaves, or the tincture or infusion of digitalis, rather than that of the glucosides derived from the plant. We know fairly well the effects of the whole drug, but there is still much dispute as to the effects, and even as to the exact composition, of some of its derivatives. I nearly lost a patient some years ago, from sudden cardiac failure, from permitting him to continue with some form of digitalin prescribed by his former medical attendant in the East. He lost his cardiac compensation while taking it. The use of the digitalin was stopped at once, and half a drachm of the tincture given, and smaller doses given afterwards. In a few hours his prostration disappeared, the functional leakage at his mitral orifice disappeared, the cardiac dullness returned to nearly its normal area, having been much enlarged by acute dilatation, and under continued use of this preparation the

patient continued in comfort for two years. I have never yet had reason to doubt the reliability of a well-made preparation of digitalis, whether prescribing the tincture or the infusion. Unless previously tested, I cannot believe that the various digitalins on the market should be used in grave cases, for we then need a drug upon which we may absolutely depend. I will not here enter into the discussion of the relative merits of the infusion and the tincture, for it is to be hoped that the indications for these preparations will be more thoroughly worked out within a few years. There is reason for hope, too, that we shall soon possess derivatives of digitalis which may replace, without sacrifice of efficiency and reliability, the preparations just mentioned.

# Treatment.

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## THE TREATMENT OF TUBERCULOSIS.

BY PROFESSOR GRANCHER,

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### LECTURE I.

GENTLEMEN,—I propose to deliver a series of clinics on the treatment of tuberculosis, and I shall limit myself practically to pulmonary tuberculosis, because this is by far the commonest form, and because it is also the form that kills so many patients,—often, I believe, through the physician's fault, or, to be more just, through the fault of the education that physicians receive. I shall first show you that neither physician, patient, nor the patient's family are prepared for the hygienic treatment, the only efficacious one of tuberculosis.

What does a physician see and learn while studying his profession in our hospitals?

It is very rare for him to see in these hospitals cases of pulmonary tuberculosis in the early stage, where an early diagnosis has been made, and which are treated in the proper manner,—that is to say, with energy and for a long period. He rarely sees them because, on the one hand, patients but slightly affected do not come to the hospital, or if they do come, complain of something else than their lung trouble and are classed under another heading, such as chlorosis or anæmia; when these patients complain of their cough, superficial auscultation fails to detect the gravity of the complaint. Again, when the hospital physician makes an early and exact diagnosis, and wishes to prescribe the necessary treatment and to carry it through successfully, he is prevented by our defective hospitals, which do not give to such patients the diet or surroundings they require. If he overcomes, even in part, these difficulties, he is ham-



pered by the patient, who, feeling much better after a few weeks of treatment, thinks that the trouble is entirely cured, and leaves the hospital in spite of any advice to the contrary.

Consequently, a medical student rarely sees the instructive and encouraging sight of a tubercular patient cured by a rational, hygienic treatment that aims at restoring the strength of the organism, and by so doing indirectly succeeds in curing the tubercles.

What the student sees and learns is just the contrary. The tubercular patient who remains in the hospital is the consumptive who has reached the period of breaking down, or excavation, with fever, night-sweats, vomiting, etc. This wretched being has generally lost all appetite, and the food that is brought to his bed, although of good quality, is quite insufficient (even when raw, scraped meat is added to it) to effect the cure of phthisis.

It should also be remembered that the atmosphere of a hospital ward, where one patient out of three is consumptive, and where the windows are generally closed both day and night, is not suitable either for a consumptive or any one else. As for rest, so indispensable for this class of sick persons, the activity in the ward steadily deprives them of it. In the daytime nurses, physicians, and visitors go and come, while the ward is swept, dusted, and the curtains shaken in the same way that it was done twenty years ago. In the night, the neighboring patient coughs or groans, while the consumptive, kept awake by these noises, and also by his own coughing, has no chance to sleep himself, and prevents his neighbors from doing so as well.

Consequently, the physician, however energetic and learned he may be, gives up the hopeless struggle, and his visit to the consumptive's bedside is a mere form: he changes one quieting remedy for another, says a few kind words of encouragement, and passes on. The patient soon realizes the situation, and either leaves the hospital to seek elsewhere a recovery that he will not find, or else becomes resigned and waits for the end. As for the medical student, he naturally carries away (and keeps) the conviction that phthisis is incurable.

In view of these facts it is not astonishing that so many physicians, who after all cannot be expected to know more than what they have seen and heard, should be badly prepared for the difficult and complex problem they have to solve: of making the diagnosis of tuberculosis in its earliest stages, and in attempting to cure it.

Their preparation will seem still more incomplete if we admit that there is another thing difficult to learn in a hospital: the art of taking care of a weak, delicate stomach, of putting it into shape again, and of restoring its strength and vigor. This art is an impossible one to learn in our city hospitals, owing to the confined life in an unhealthy atmosphere, and to the necessarily rudimentary and uniform preparation of the food, as tubercular dyspeptics, more than all others, require pure air and choice food.

Consequently, a young physician leaves the school of medicine very ignorant, in most cases, of the many resources at his disposal to cure tuberculosis. Little by little, if he uses his eyes and if he reads, he will learn what he does not yet know; but he will do so slowly and to the detriment of many patients to whom he will not have been as serviceable as he might have been.

On the other hand, the patient is no better prepared than the physician for this hygienic treatment. When he comes to see his physician for a "neglected cold," he has no suspicion of his serious condition, but thinks that a few days of rest and a good prescription will restore to him what he has been losing for weeks, months, or years,—that is to say, appetite, flesh, and strength. This patient, whom I suppose to be on the threshold of phthisis, or in what is usually known as the first period, has been losing flesh and color for a long time, and his stomach is in poor order; these points, however, have not attracted his attention, and he does not mention them until the close questioning of the physician reveals to him the progressive loss of condition that preceded, accompanied, or followed his cough. The only symptom that troubles these patients is their cough, which is certainly the least important of all.

In other cases, but less frequently, the patient comes trembling and frightened. He has met a friend and has been told that he looks badly, seems to cough a good deal, and ought to see his physician. Since every one has heard phthisis spoken of, has seen a relative, friend, or neighbor die of it, and lives in dread of it for himself, he comes to get an opinion on his condition and to know what to do.

But whether he be frightened or confident the tubercular patient expects to be quickly and easily cured. He also expects to be cured by a remedy or prescription, and his physician will be only too tempted to give him one, as he is constantly reading in the medical papers of this or that remedy that is sure to cure tuberculosis in its

early stages. It is not necessary to say that this remedy is constantly varying, and that each successive one only keeps up its reputation for a short time. The physician, therefore, prescribes the remedy of the day, and since at the same time the patient takes a certain number of precautions, the improvement which almost always follows this first visit deceives everybody. But it does not last long, and soon the first drug has to be changed for another, and then for another, and the patient steadily loses ground. The patient then begins to go from one physician to another until his case becomes hopeless; nor does this take long, as the hours are precious and each week lost in looking for a remedy that will cure, but which unfortunately does not exist, will have to be redeemed by long months of steady effort, if it is not already too late.

Since the serum treatment has come to the front, the patient, who has heard of wonders realized and of hopes raised on all sides, asks to be treated with the serum. This is done, and the natural serums of the dog, goat, or sheep, the artificial serums or saline solutions, and the therapeutic serums of Koch or Maragliano, are used and without success.

Experience has shown that as soon as the period of suggestion is over this treatment is always useless and always dangerous, and I cannot help wondering at the readiness with which physicians and patients allow themselves to be drawn into these risky experiments. How many times of late years have I been obliged to defend myself against the prayers and entreaties of patients and their physicians! I can understand this eagerness on the part of the patient, who is absolutely determined to recover; what I understand less is the readiness with which certain physicians, well-known men whose situation ought to teach them greater prudence, hold forth hopes of recovery based on a few hasty and inconclusive experiments.

Although I am one of those who, in spite of the failure in the attempts that have been made so far, hope that vaccination and immunity for the tubercular bacillus will some day be found, I do not deceive myself as to the value of this future discovery. A serum of this sort, if it is harmless, and can be used preventively, will be able to protect a healthy person for a certain length of time against tuberculosis; but the sick, and there will always be such, will not be able to count on really recovering, even if the serum destroys the bacilli, unless they follow for a long time a course of hygienic thera-

peutics. The latter means alone is capable of repairing and healing lesions, of restoring strength and tissue, and of equalizing the receipts and expenses of the organism. This equilibrium is nothing else than the definition of health,—that is to say, of recovery. Whatever else may be done, we shall, therefore, always be obliged to apply the treatment by food, fresh air, and sunshine, without which our cells cannot make good their losses. The serum treatment will be able to do nothing more than to neutralize the baneful action of the tuberculine on our tissues; it will be powerless to create either a red or a white globule. The latter, which are the most active agents of the cellular life of our organs, and are the true factors of recovery, we create with food and good assimilation.

Let us, therefore, begin by using this treatment, which will always be necessary both for tubercular and other bacilli, since, after all, no one may find the serum in question, and since we have the very best reasons for knowing that this hygienic treatment, which is always useful, is able by itself to cure tuberculosis.

We have seen that the patients, who all expect to recover quickly, think that it can only be done with the help of the druggist or laboratory. When these patients are anxious they are afraid to learn the truth about their condition, and either say so themselves or get some one to say it for them. Even when they ask to have the whole truth told them, their attitude and language show very plainly that they do not wish to be believed. It is rare to see a patient admit that he has lost a good deal of flesh, that he has not been well for a long time past, and that his strength has seriously diminished; it is usually the person who accompanies him, mother, sister, or wife, who corrects his replies and enlightens the physician.

But it also often happens that the physician is as much disinclined to tell the patient the truth as the latter is to know it, especially if he is a new arrival in the city in which he practises and has not yet established a reputation, or if he is on social terms with his patient and takes into consideration what the family is going to think. Consequently, through a natural horror of tuberculosis, and also through the curious frame of mind owing to which it is difficult for a human being to admit that he is seriously affected, the patient shuns the truth; while the physician, through pity or weakness, hides it. On the other hand, the family, who pretend to be anxious to know the truth, are delighted if the physician holds out hope for

a speedy recovery. It is difficult to refuse such hope to a distressed mother or father. Consequently, in this first decisive interview with the physician, on which the life of the patient may depend, everything tends to deceive both patient and physician, who are both led to try the wrong path.

When, on the other hand, the physician is an exception to the rule, has but slight confidence in new remedies, and intends to make hygiene the basis of his treatment,—when he has the skill to make an early diagnosis,—when he has a strong character and his mind made up that in order to get his patient to obey he must make him understand the seriousness of the situation, he quickly finds that there is an abyss between himself and his patient. The family, also, by instinct take the patient's side: "What, consumptive? there are none in our family!" and this fine reason, or, I should say, nonsense, will suffice to take them to another physician, who will call it bronchitis and give a prescription.

To avoid all these difficulties, including that of the loss of a patient, I readily admit that a good physician has to be as well a psychologist and a diplomatist. In the patient's interest he must tell him as much of the truth as is necessary for the treatment, but no more. He need not be severe, or pessimistic, and still less brutal; but must with skilful and prudent firmness choose his expressions and measure the amount of plain language that the patient will be likely to support. He must know how to temporize, if necessary, and postpone to a second visit the inevitable explanation, when there is reason to dread the moral effect of a too sudden disclosure. It is sometimes even necessary to adopt the patient's line of thought, and to give a prescription, as there are some that are harmless and even useful; but the great and most important point is to begin the real treatment without losing an hour.

Here the difficulties begin to arise, as this treatment cannot be indicated in a few lines. It is impossible for a physician to show too much care, tact, or authority in going into the most minute details concerning everything that a tubercular patient must do to recover. The general indication is always the same: to fortify the body by alimentary and respiratory hygiene; but each patient requires to be convinced by his physician, and to have his mind turned towards this therapeutical method, which appears so simple and unpretending that it is at first difficult for him to believe in its

efficacy. He argues that if to recover all you have to do is to eat, digest, and breathe properly, why is it that he is ill, as he does all that already! It is necessary by a skilful and detailed cross-examination to prove to him in the first place that his disease dates farther back than his cough; that he has lost sight of former illnesses that bear on the question: pleurisy, bronchitis, or hæmoptysis; that his strength has been decreasing for a long time; that his persistent cough is only a tardy manifestation of the disease, which is characterized particularly by his loss of strength, pallor, and his decrease in weight; that that is his real disease, and not his cough. The digestive functions must be very closely examined, and the faults committed in the matter of diet (and they are always numerous) detected, so as to show him why and how his digestion is poor or bad. He must also be taught that to breathe vitiated or confined air is bad for his disease, and he must be shown how to take into his lung a large amount of pure air. In a word, the physician must put his patient through a course of instruction on all these points, as he will be certain to be found ignorant in all respects concerning his diet, how to breathe, and the necessity of taking proper rest. Finally, the physician must upset his prejudices and blind confidence in the omnipotence of medicines, which must be put in their proper place in the second rank.

I admit that this is not an easy matter, especially for a young physician, but it becomes relatively easy for any one who is convinced of the value of the hygienic treatment of tuberculosis. A strong conviction gives the necessary authority to speak clearly, to point out the peril, and to ward it off. But conviction, even based on true knowledge, is not all that is required; great skill is necessary to know what to say and how to say it. No two of these patients are alike, by which I mean that, although their lungs may be in the same condition as regards the disease, their stomachs will be very different, and still more their social and mental conditions.

There is, therefore, no advice that requires more time and more tact to give than that which a physician owes to a tubercular patient who ought to get well. By this I mean a patient whose disease is only beginning, and who is willing and able to use every effort and make every sacrifice for recovery.

There are some tubercular patients who ought to recover, while others cannot do so. If we take a group who are as nearly as pos-

sible alike in pulmonary lesions and general condition, their lot will depend on their intelligence, docility, and will, and also (unfortunately) on their pecuniary resources. The most important requisite for recovery from this lung-disease is to be able to give up everything and rest for several months at least, and to afford material comforts that are expensive, such as: copious, choice food; sunny, healthy dwellings; and proper clothing, etc.

Although actual wealth is not necessary, such patients have to be in easy circumstances and in a position to lay aside for some time the cares of life. This, of course, for many is an impossibility.

In behalf of this large class of patients our public hospitals must change their methods entirely, and create sanatoria, not for consumptives with cavities, as these unhappy patients are practically doomed, and society can only furnish them with a hospital where they can end their days in peace, but for tubercular patients in the extreme early stages, where a good diagnosis of probability has been made.

Such patients react wonderfully to a hygienic treatment; in a few months they can no longer be recognized, have recovered their strength, and are able to return to work, if, having learned to take care of themselves, they are sufficiently wise and energetic to do so.

As regards the class who are in easy circumstances or rich, and who are able to treat themselves, I will first say that it does not suffice to be able, but that they must also be willing to take care of themselves; they must help the physician and consent to be ill,—that is to say, be satisfied with a quiet and prudent existence, from which the noisy and dangerous pleasures of social life are carefully excluded, and in which family life, carefully regulated, must fill all desires and be sufficient. How few of these patients consent to a monotonous existence in which books and home life replace for several years theatres, dinners, balls, and clubs! Yet this is absolutely necessary for recovery, and if you do not begin by convincing your patients of this fact, the benefits of your advice will be neutralized as rapidly as they are obtained.

It is, therefore, not sufficient for you to ascertain while talking with your patient the probable nature of his disease; you must then find out the state of his fortune, what his family consists of, and his mental frame of mind, in order to know what you are to tell him, and how you are to do so, in order to bring him to the required

point of obedience and faith to get him to follow the treatment for a long period and without relaxation.

You will find patients intelligent enough and with sufficient moral energy to understand you at once and to follow your advice with scrupulous care. These are the good patients, whom you have frightened to the right point, who have confidence in you, and who have in themselves sufficient determination to carry out a long and tedious treatment.

I often say that to be sure of recovering from pulmonary tuberculosis you have to be of easy means, to be afraid of death, to love life sufficiently to make any sacrifice, to understand the spirit more than the letter of your physician's advice, and, finally, to ask or require your family to help in the work of recovery. This ideal type is not often met with, but still it is found sometimes even with certain of these qualities exaggerated, especially the selfishness.

A few years ago I met in a winter resort a young man very ill but very intelligent and very desirous to recover, who was following out his treatment under the advice of a very capable physician. We became acquainted, and he was fond of talking to me of his troubles, listening attentively to what I had to say, and, as he afterwards confessed, putting my advice immediately into practice. My remarks only corroborated those of his physician, but he used them in disciplining his entire family, mother, wife, and children, and in breaking in everybody around him to his diet, life, and exercise. It did them all good in the end, but at first they only obeyed through devotion and submission; in this way he carried out his treatment with his family with the least amount of boredom and the greatest amount of amusement possible. He recovered quickly and thoroughly; I saw him a few years later, and again quite recently, when he had taken up his usual life after four years' interruption.

Happier are those who have about them an affectionate family, whose members support each other mutually, and are prepared for any sacrifice to save the one who is in danger. How much easier is the victory when the patient has some one near him to encourage and sustain him in the struggle against tuberculosis! How inestimably precious is the collaboration of an attentive mother or wife, always there to remind him of his orders, to see that they are carried out, to cheer him up in moments of discouragement, to show him



the danger, especially when, over-confident in his improvement, he thinks he is cured and is inclined to be imprudent again!

Unfortunately, it is the contrary that usually happens, and the patient is isolated or abandoned, either because he has no relatives, or because they are not able or willing to devote themselves to him. Recovery is then far less probable, especially in the case of a young person. In many cases there is no lack of affection, but insufficiency of purpose and foolish belief in anything that is heard. In this way a mother, though full of tenderness for her child, may make a wretched nurse on account of her love, through lack of energy or authority, and also through ignorance. I am, therefore, very prone in the case of tubercular patients to advise physicians to look out for the persons about the patients, as unconsciously they may be the cause of a great deal of trouble.

Let me mention some other types of good patients for your edification.

Mme. X. is a very intelligent young Parisian, very fond of society, but in delicate health for some time past. I saw her in 1885 for the first time, seriously ill, very thin, with fever, hæmoptysis, and infiltration of the entire left lung. In spite of this she recovered so completely that, after spending three winters at Mentone, she went back to her former mode of life, the country near Paris in summer, and Paris itself in winter, as her husband cannot get away. To do this she regulated her whole life herself, watching her symptoms with great skill, and taking particular care to avoid any cause for a relapse. One detail will show what the woman is. She has a reception day in winter, but does not accompany anybody to the door, nor does she ever leave her place in the parlor; when she is alone she paints and reads and is never bored. In the mean time she has carried through a pregnancy without trouble, and her child, a fine boy, is now two years old. She never has any appetite, and her stomach is dilated and in poor condition; yet she makes the best possible use of it, eats and digests her food, in spite of these facts, by sheer will-power, prudence, and care in her diet. To me this woman is a most striking example of what can be done by a person determined to get well in spite of a weak and already deteriorated organism.

A physician related to me the following typical case. He has an uncle, now eighty-two years of age, who lives on a place in the

country, where he still hunts and is very robust. Sixty years ago, when this uncle was a medical student, he began to lose flesh, cough constantly, suffer from a hectic temperature, and he had a number of hemorrhages. His physician told him to leave Paris and his studies and to live in the country like a peasant. To stop his hemorrhages and to prevent their return he was not to speak above a whisper. He followed this advice so scrupulously that although his hemorrhages had long ceased he still continued to speak in a whisper, and for three years no one heard the sound of his voice. Ten years later, in his thirty-second year, he married and had four sons, who died successively of tuberculosis when they were about twenty. The mother died of grief, but he continued in good health. You will have no trouble in imagining what an energetic man, as well as selfish, no doubt, this patient must be who condemned himself to silence for three years, and how useful to him must have been his fear of his hemorrhages and of death. The loss of all his sons is due, I imagine, to the fact that they were less anxious to live than their father, or that they had not the energy or will to pay for it so dearly.

There are other good patients, even though they lack in energy, because they listen to a will-power stronger than theirs, such as Mme. de S., whose history I published elsewhere, and who was actually *compelled* during several months by her sister to take the food that had been prescribed. In other cases they are children, or docile young people, who obey their parents. In a word, from whatever source the determination to get well may come, it must be constantly present.

On the other hand, how many are wretched patients! I am only speaking of those who might get well and will not do it.

I am taking care of a young lady of twenty-three with another physician. I knew her as a child, but when she was brought to me in July, 1895, I found that she had tuberculosis in an active form. She suspected this, but would neither obey her physician, an old friend, afraid to lay down the law, nor her father and mother, whom in fact she had never obeyed. Knowing the patient and her surroundings, I intervened with all the energy I possessed. I persuaded her to take care of herself, and she kept her word until the end of October. She then came to see me with her physician and mother, and as I was congratulating her on the improvement in her health (she had gained thirteen pounds, stopped coughing, and

was breathing much more freely) she said, in very short tones, "That is all very well; but I have been taking care of myself for one hundred and twenty-five days now, and I am tired of it; I would rather have a good time for a while and then die, if necessary." I saw, however, by the way in which she said this that she did not half mean it, and without much trouble I obtained her promise to go on with the treatment for another period. I doubt whether this case will be a successful one, as she is so spoiled by her parents that she rules them absolutely. As for the physician, a very capable man, he has no authority, owing to his situation as a friend of the family.

A young man came to see me awhile ago in the country with all the signs of a rapid form of tuberculosis with hectic temperature, whereas I had hoped to find marked improvement, as I had seen the patient two months before and he had promised to follow my advice. There was no lack of intelligence on his part, but as I was told by one of his friends who came with him, no one could expect any improvement in his case with the life he was living, never home before three in the morning! When I asked him what he was doing out at such hours as that, he replied that he was with his friends, but that he committed no excesses, and only drank milk. This he called treating himself. Three months later he was dead.

Last year a father brought his son to see me, a tall, slender young man, in full febrile tuberculosis. I tried to hammer into the mind of this unfortunate youth the necessity for a long and energetic treatment by rest, fresh air, and food, but his reply was, "Then there is to be no more horseback-riding, bicycling, or hunting?" "No," said I; "but you will have other occupations; you can read, or draw, or anything you like of that sort." His only answer was that he was bored to death by everything of that kind, and nothing further than that could we get out of him. He died in a few months, another example of a child thoroughly spoiled by indulgent parents.

Mlle. X., very well known in Paris on account of her beauty and talent, came to see me twice with her physician. The case was so far advanced that the situation was desperate; but still I wished to make an attempt. I found myself, however, absolutely frustrated by her determination to go on with her work as a sculptor. "Life,"

she said, "is not worth an artistic sensation," and she continued at her usual occupation until she died.

The conclusion to be drawn from these remarks is that although tuberculosis often gets well of its own accord, still, when it has caused pulmonary lesions represented by the classical signs of the first period, it requires a long and monotonous treatment. Therefore, in order to get well, a thorough determination to do so is the first of all requisites.

## REMEDIAL MEASURES IN OBSTRUCTION OF THE COMMON BILE-DUCT.

PAPER READ BEFORE THE AMERICAN SURGICAL ASSOCIATION.

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GENTLEMEN,—A paper upon the surgery of the gall-bladder and ducts was presented to the Southern Surgical and Gynæcological Association at its recent meeting in St. Louis, and appears in the Transactions of that body. As a very full abstract of that article has been published in a number of medical journals, thus affording an opportunity for the Fellows of this Association to learn its contents, I may appropriately confine my attention on this occasion to the troubles growing out of obstruction to the common bile-duct.

It is not my purpose to consider the results of pressure upon the duct from tumors and growths outside of it, but to limit my investigation to inflammatory processes involving the coats of the common duct and to mechanical impediments to the free outlet of the bile into the duodenum.

There are certain spasmodic conditions, known as biliary colic, which are generally attributed to the passage of gall-stones, and which cease so soon as the calculus reaches the duodenum. It is evident also that many lesser derangements are connected with greater or less constriction of the common bile-duct than that which is characterized as catarrhal inflammation of its walls, doubtless in some instances from spasmodic conditions of the duct independent of the presence of calculi in the canal.

The cases of jaundice which result from the temporary closure or constriction of the common duct are usually left to the treatment

• of the physician, and yet are the precursors of derangements which require surgical interference, and hence come within the sphere of this paper.

They commence with functional disturbance but terminate in organic derangement. Murphy lays great stress upon the relief of jaundice by pilocarpine as a means of distinguishing catarrhal obstruction from that caused by biliary calculi, and claims that the catarrhal inflammation is uniformly controlled by this method of treatment. In his article on "Obstruction of the Ductus Choledochus," in the "Twentieth Century Practice of Medicine," page 761, he says,—

"To differentiate mechanical from catarrhal jaundice, it has been suggested that one-sixtieth of a grain of hydrochlorate of pilocarpine be given twice daily for two weeks."

If I am not mistaken, the reliance of the greater number of practitioners for relief in this class of cases has been, and continues to be, upon the phosphate of soda.

A very convenient form of dispensing this remedy is in solution, and has been employed with good effects in temporary interruption of the flow of bile from the gall-bladder, and may be given for several days with advantage.

In dealing with the formation of gall-stones, impacted in the common bile-duct, as well as collections in the gall-bladder, various medicines have been resorted to with benefit; but there have been more good results claimed from the employment of olive oil internally than from any other remedy of this class. There may have been some misapprehension in regard to the formation of spherical masses of fatty deposits with cholesterin in the evacuations from the bowels after taking large doses of olive oil. These balls are sometimes so solid as to be taken for calculi, but are readily crushed by pressure and do not contain the elements of gall-stones.

But genuine biliary calculi are often expelled by olive oil, and sometimes relief is permanent from this treatment.

I have resorted to this mode of relief in a considerable number of cases. Even when no gall-stones were discovered, there has been decided benefit from taking a pint of olive oil in four doses within twenty-four hours, and afterwards two ounces night and morning for a week, with the use of an ounce at night for another week or two. This course of treatment has been followed by the use of

R Fluid extract of taraxacum, fʒi;  
Dilute muriatic acid, fʒss;  
Fairchild's essence pepsin, fʒiv;

in teaspoonful doses three times a day with a little water.

Such a combination improves the digestion by acting upon the liver and obviates biliary complications. So far as these measures effect any modifications of the structure, they may be recognized as surgical, and I would not exclude all medicinal treatment from the appliances which belong to the domain of surgery. We should not view operative procedures as the only recourse of the surgeon, and while mere functional derangements pertain to the physician, those changes of the tissues which tend to organic or structural derangements belong to the sphere of the surgeon. The steps to be taken as prophylactic means or the measures adapted as preparatory to operative work are to be considered as surgical in their practical bearing upon the result of the case.

After exhausting medico-chirurgical means of treatment for biliary complications, without arresting obstruction of the common bile-duct, the surgeon is confronted with conditions calling for operative interference. There may exist a stenosis or stricture of the walls of the duct, without occlusion of the canal, dependent upon inflammation; or there may be a partial obstruction of the lumen of the duct from the presence of a gall-stone, which permits the bile to pass around it to a limited extent. In some cases Fenger has demonstrated that a gall-stone may be so located as to form a valvular closure at times, and to drop back at other times into a larger portion of the duct, so as to leave a freer outlet for the bile into the duodenum, thus accounting for alternations in the appearance of jaundice.

The entire occlusion of the lumen of the common bile-duct, whether from adhesive inflammation of its walls or by the presence of biliary calculi in the canal, is ordinarily attended with the absorption of bile into the general system and the absence of any bile in the intestinal canal. Under such conditions, the fecal evacuations are without the usual coloring matter of the bile and present a light clay color, while the urine has a deep tinge of bile and is sometimes of a dark green or yellow hue.

The yellow tinge of the skin generally passes gradually into a deep bronze color, accompanied with great itching of the entire

surface of the body. Digestion is much impaired, and cholæmia is marked by great lassitude and general vital depression, with despondency and mental hebetude.

The absence of the bile from the alimentary canal and the consequent deprivation of it in the digestive process accounts for the impairment of nutrition. But the further interference with assimilation is evident from the presence of bile in the various tissues, by absorption through the lymphatics, constituting cholæmia. The toxic properties of the bile when it is arrested in the ducts and gall-bladder, by an impediment in the common duct, are not so evident as when it enters the circulation and develops jaundice. Though an outlet may be artificially provided for the bile by connecting an opening in the gall-bladder with one in the abdominal wall, there is not always relief to the cholæmia, and its effects upon the general health of the patient continue, even to a fatal termination. It is the duty, therefore, of the surgeon to anticipate the grave consequences of obstruction of the common bile-duct by a timely resort to means of relief. In undertaking to make a diagnosis of the source of trouble in cases of the arrest of the flow of bile into the duodenum, the resort to an exploratory operation is often necessary, and the indication for this is generally clear by the presentation of a protuberance of the distended gall-bladder with or without enlargement and induration of the liver. The incision which is usually preferred extends from the outer margin of the right rectus muscle backward below the edge of the liver, parallel with the border of the costal cartilages and at a distance from this line, depending upon the descent of the liver below the ribs.

In a case presenting no enlargement of the liver the incision should be made on a line an inch and a half below the border of the costal cartilages, and should be about three inches in length through all the tissues of the abdominal wall. If the conditions of the structures require more space for examination, this incision may be extended at either end, and very rarely, if ever, is anything gained by a longitudinal incision in the line of the rectus muscle as practised by some operators.

With this oblique opening into the abdominal cavity, the surgeon is enabled to explore all the region involved in trouble from obstruction of the common bile-duct, and to accomplish any operative procedure that may be required upon the ducts or upon the



gall-bladder. The hand can be passed within this opening for palpation of the viscera, and most of the structures can be seen by the eye.

No reference is requisite to the mode of exploration by a trocar, which has been used by some operators for detecting stones in the gall-bladder or for drawing off its fluid contents, preparatory to other operative procedures. I have elsewhere given an unfavorable opinion in regard to this exploratory puncture, and do not consider it entitled to recognition as a means of diagnosis or measure of treatment. An exploratory operation by a free incision may avoid the risks of such a puncture, and affords better facilities for information as to the further radical measures to be adopted in the case. Palpation of the gall-bladder and inspection of it will guide the operator as to evacuating its contents, while manipulation along the cystic and common ducts will detect any obstruction by biliary calculi or the agglutination of their walls by inflammation or distention by inspissated bile. This incision enabled me on one occasion to pass the hand within the abdomen and to lift the gall-bladder from the iliac fossa out of the opening, and on the same occasion to verify, by palpation from the other end of the incision, the occlusion of the common bile-duct near its entrance into the duodenum. It has the further advantage, by cutting through the tissues obliquely, of securing a better result from suture when closed, and thus being less liable to hernial protrusions than an incision in the *linea alba* or the *linea semilunaris*. I am thus emphatic in preferring the oblique line below the costal cartilages in exploratory and other operations for trouble connected with obstruction of the bile-ducts from observation of its advantages over other modes of procedure adopted by some distinguished operators in this branch of surgery, and consider it essential for the best result.

The various measures recognized for the relief of obstruction of the common duct by gall-stones are breaking up of the calculi by the needle, crushing by padded forceps, forcing them into the gall-bladder or duodenum, catheterization, and excision through the walls of the duct.

Traction upon a calculus so as to bring it backward into the gall-bladder, from which it is to be removed by incision, is a procedure to be commended in cases when the stone is located near the opening of the cystic duct.

In the event none of these procedures prove successful in removing the impediment to the flow of bile into the duodenum, or it is found that impermeable agglutination of the walls of the common duct exists as a barrier to the biliary discharge by the natural channel, it then becomes requisite to provide an artificial outlet of the bile from the gall-bladder. In some cases it is impracticable to attach the gall-bladder to the parietal opening or to suture the incision in the wall of the duct when a stone is extracted, and thus it becomes necessary to provide for drainage by packing with gauze, around the field of operation, and leave the ends of the strips extending out of the wound.

It has been shown by Dr. W. E. B. Davis that this procedure proved satisfactory in his experiments upon dogs, and operators have had occasion to verify the advantages of this practice upon the human being when it was impracticable to effect drainage otherwise. While freshly secreted bile from a healthy liver has no septic property, there is evidently a harmful effect of vitiated bile from a diseased liver, and hence it is desirable to carry it off by drainage.

In doing operations upon three white women for obstruction of the common bile-duct, by forming a fistulous opening for the bile externally, the cholæmia persisted in an aggravated form until death supervened in each case. There was a free discharge of bile through the external orifice, but this did not afford relief to the dyscrasia and toxæmia resulting from the absorption of vitiated bile previously. It is true that a slight intoxication of jaundice from temporary obstruction of the common duct may disappear under medicinal treatment, and even profound cholæmia of short duration may be relieved by giving an external exit to the bile, but when the bile permeates the tissues and remains for a long time it becomes a poison to the system.

In some conditions of temporary impediment to the flow of bile through the duct into the duodenum, the attachment of the incised gall-bladder to the parietal opening has relieved the obstruction and been followed by the restoration of the bile to its natural channel.

It is held by some that, even when the occlusion is persistent, the escape of the bile by an external fistulous opening entails no serious consequences upon the health of the patient. But the bad result of the deprivation of the bile from the alimentary canal is a matter of common observation, and this condition of an external fistulous discharge is itself an urgent ground for operative inter-

ference to return the bile to the alimentary canal. As there seems to have been misapprehension on the part of some surgeons as to the claims of this measure of relief in obstruction of the common bile-duct, it is in order to state that only in complete occlusion of the duct, which has proved intractable to other procedures, has an artificial opening from the gall-bladder or the common bile-duct into the alimentary canal been thought proper.

It is begging the question to allege that any of the devices resorted to for the removal of stone or for the dilatation of the stenosis of the duct, with or without an external discharge of the bile through a fistulous opening, can take the place of a radical means of relief by attaching the gall-bladder to the duodenum or a coil of the small intestine. It is in cases not admitting of another recourse that cholecystenterostomy is indicated as a radical operation.

This anastomosis is effected in various ways, either with the duodenum, the small intestine, or the colon, the last named being least desirable, as it fails to secure the advantages of the bile in the process of intestinal digestion. My experiments upon dogs demonstrated the feasibility of this fistulous communication of the gall-bladder with the bowels, and it is a matter of minor importance by what process it is accomplished. It is no part of my undertaking to set forth any special technique for securing the end in view of connecting the gall-bladder or the common duct with the alimentary canal so as to admit of the passage of the bile into the latter. My own aim has been to place the operation of cholecystenterostomy on the basis of a life-saving operation, when all other means are found to be fruitless in cases of complete occlusion of the common bile-duct.

Some prominent surgeons, and most conspicuously Tait, of Birmingham, have ignored or condemned this radical means of relief, but a sufficient number of successful results have crowned the operations of equally distinguished members of the profession to offset any mere speculative opinions adverse to it. If I have been instrumental in directing attention to this procedure as the sole agency in correcting an otherwise irremediable condition of things connected with occlusion of the common bile-duct, I ought to rest satisfied with the result.

The statistics in the past show that the various modes of procedure are comparatively free from danger and are attended with a marked degree of success, so that we may reasonably expect cho-

lecystenterostomy to be recognized as a safe and efficacious recourse in the future annals of surgery.

From the foregoing considerations we make the following inferences:

1. That obstruction of the common bile-duct may result to a greater or less extent from catarrhal inflammation or from gall-stones.

2. The ordinary accompaniment of any obstruction of the common bile-duct is jaundice and the absence of the usual color in the fecal evacuations.

3. In the lesser developments of jaundice medication is to be relied on for relief, but in the more accentuated and persistent forms operative measures are always requisite.

4. An exploratory operation by incision diagonally beneath the right costal cartilages enables the operator to determine upon the nature of the impediment and the best means of relief.

5. In the various cases we may resort to catheterization, needling, crushing, traction, incision of duct or gall-bladder, with attachment of either to the parietes or intestine.

6. That complete occlusion of the common duct cannot be remedied by the outlet of bile through a parietal opening, but calls for a communication of the gall-bladder or the duct with the intestinal canal.

7. The duodenum affords most favorable conditions for this connection, a coil of small intestine comes next in its advantage, and the colon presents least benefits, as it fails to obtain the good effects of the bile for intestinal digestion.

8. The anastomosis of the biliary system with the alimentary canal may be accomplished by different processes, and the selection of the mode best adapted to the case may be left to the choice of the operator.

9. Permanent occlusion of the common duct associated with a patulous cystic duct is the only condition in which cholecystenterostomy is indicated, and for this condition of things it may be considered a radical mode of relief.

10. While a fistulous communication of the gall-bladder or common duct with the alimentary canal is a safe and efficacious mode of relief for all ordinary results of occlusion, it does not afford exemption from the consequences of malignant growth, and hence this kind of obstruction is not included in this paper.

## THE TREATMENT OF ACUTE FAILURE IN CHRONIC HEART-DISEASE.

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By the term "cardiac failure" is meant an incapacity of the heart to perform the work required of it,—an incapacity arising from any cause. Too often the term is associated with valvular disease, and murmurs are sought for as the evidence of heart-disease to the exclusion of other and more important phenomena. This is a matter requiring to be emphasized, because the decision of the question of the existence of disease of the heart, its character, extent, and gravity, is so often based on the presence of a murmur and its character.

The valves are but mechanical structures, and disease of them, apart from infective processes, gives rise to symptoms only as it deranges their mechanical action and thus leads to disturbance of the functions of the heart, and through this to degeneration of the cardiac walls. Cases resulting from lesions of the valves form only a small proportion of all the cases of heart-failure.

Pathological changes in the heart occur much more frequently as part of disease of the general vascular system. It is in such cases that the most mature judgment is required in estimating the amount of injury to the cardiac muscle and its powers of recuperation, as well as the best means to be adopted to restore its powers so that they may be adequate to the demands made upon them.

In the treatment of cardiac failure our aim should be twofold,—viz., first to reduce the work of the heart as much as possible, and, secondly, to stimulate and strengthen its walls so as to enable them to perform their work adequately.

Of the means at our disposal to reduce the demand made on the heart the most important is, of course, rest. By rest we seek to relieve the heart of all work except that necessary purely for the main-

tenance of life. Many patients are restored again and again from incompetency of the heart by rest in bed alone. Each recurring attack of failure is more easily induced than its predecessor, and the restoration of competence is effected with more difficulty, until rest alone is unequal to the task of restoring effective power to the heart.

As the effect of physical labor is to greatly increase the strain on the heart, it follows that the benefit of rest in restoring the failing heart will be much greater in the man engaged in physical labor than in the man leading an easy life. In the laborer the heart obtains relief to a much greater degree by rest than does the heart of his more fortunate neighbor. In the former, rest may mean the removal of three-fourths or more of the heart's ordinary labor, while in the latter, not more than one-fourth or even less of its work is removed. Another fact of great importance in the difference between the two is that in the laboring man the diseased heart fails early, while in the man of easy life it is not until the heart is far advanced in degeneration that he shows the signs of failure. The former will therefore possess much greater recuperative power than the latter.

When the cardiac changes have advanced so far that rest alone is insufficient to restore competency to the heart's action, the end, for a time, may be attained by the use of cardiac tonics and stimulants to aid the rest in restoring the equilibrium.

In addition to rest the labor of the heart is also greatly relieved by full elimination of the waste products in the blood through the kidneys and bowels. It is probable that this in a great measure affords the explanation of the great benefit often reported from the use of calomel in cardiac disease with dropsy. Any purgative that causes free liquid evacuations will do good.

If the circulation is in a fair condition, the drinking of water on an empty stomach is probably the best diuretic, and therefore the most efficient stimulant to elimination by the kidneys. Any remedy that increases the force of the circulation will, of course, proportionately increase the excretion by the kidneys.

In advanced cardiac disease, especially after repeated failures, complete rest of body and mind, aided by free administration of cardiac stimulants and tonics and the judicious use of purgatives, often fails to give relief. The signs of cardiac incompetency increase; there is increasing dropsy and dyspnoea with insomnia. The

rest of the body is not sufficient to bring the needed rest to the heart; it remains irritable; its action becomes increasingly irregular. The patient grows weaker, the paroxysmal dyspnoea increases. Almost as soon as he falls asleep a severe attack of dyspnoea causes him to awake and sit up in bed. The distress from the dyspnoea is great, but that from want of sleep is greater. These are cases really of *angina sine dolore*. Such a condition is rarely benefited by the freest possible use of digitalis or other cardiac tonics and general stimulants. If the dyspnoea is not due in part to effusion into the pleura and oedema of the lungs, a dyspnoea that is persistent and not paroxysmal as cardiac dyspnoea is, there is no remedy so effective in such a condition as morphine. The comfort afforded by a hypodermic injection is almost incredible. Given by the stomach it often fails even in much larger doses, because absorption is so slow on account of the venous stasis in the mucous membranes of the stomach as well as of other parts, caused by the impeded circulation. In most of the writings on the treatment of cardiac failure too little is made of the great benefit to be obtained by the use of morphine in such conditions. Many of them make no reference to morphine at all in connection with heart-disease; a few place its efficiency in severe heart-disease next to digitalis. I am inclined to look upon it as even more important than digitalis in these cases, in fact, as indispensable. The following cases show well its great usefulness:

CASE I.—O'Neil, aged seventy, rather a small man of spare habit. Had been a business man and not been subject to much hardship or dissipation. He was admitted to the Toronto General Hospital suffering from the effects of acute heart-failure. This was his third attack. He presented all the usual symptoms,—viz., attacks of extreme paroxysmal dyspnoea and well-marked Cheyne-Stokes respiration; insomnia, starting with an attack of dyspnoea as soon as he fell asleep; extremely irregular tumultuous action of the heart, many of the contractions giving no pulse at the wrist; considerable dropsy of the abdomen and legs; a large liver; loss of appetite with considerable flatulence. He was unable to lie down; he was propped up in bed with a back-rest. The bowels were acted on freely with calomel, followed by salines. Digitalis with general stimulants were given freely. His diet was light and nutritious. Ten days later, there being no improvement, morphine, one-fourth grain, was given subcutaneously at bedtime. It was followed by a good night's rest

with corresponding improvement in the general symptoms next day. The morphine was repeated every night for a week, by which time the heart's action was regular, the area of pericardial impact and dulness materially reduced, the dropsy had almost wholly disappeared, the appetite had become much better, and his general condition correspondingly improved. Two weeks later he left the hospital in good condition. Of course the heart was still large, and it was only a matter of time, he was told, until the symptoms would return. He was enjoined to take active exercise within the limit of perfectly easy breathing, and to avoid overstrain, exposure, over-eating, and dissipation of all kinds. Six months later he returned with a relapse, but it was much less severe than the former attack, and he recovered from it after a short stay. He has not been heard from since.

CASE II.—A Russian Jew boy, aged twelve, entered the Victoria Hospital for Sick Children March 31, 1896. He had always been a weakly child. Had a mild attack of rheumatism when nine years old. Two weeks before entrance into the hospital he had a sudden attack of hemiplegia of the right side. He had recovered the use of the leg fairly well on admission, but the arm was helpless. He complained on entrance of severe pain in the region of the spleen, for which he received morphine, one-tenth grain, three times. His heart was enormously enlarged and showed all the signs of acute failure of compensation. There appeared to be disease of both mitral and aortic valves. The ends of the fingers were clubbed to an extreme degree. A week after admission his temperature, which had reached as high as  $104^{\circ}$  F., became normal, but the circulation did not improve. The dyspnœa was very distressing, so that his nights were very restless. For this, morphine, one-eighth grain, was given subcutaneously every night with the most satisfactory results, but it had to be continued for a month. His condition improved and he was able to sit up. His temperature would frequently rise a few degrees without any apparent local cause. He did not regain strength enough to go about, but his circulation improved so much that he rested with comfort and his appetite was good. He became suddenly prostrated on June 1 and died in a few hours.

These cases serve to illustrate the utility of morphine for the relief of an extreme degree of cardiac failure. So far as my observation goes, it may be given with a free hand. I have given half a



grain without injury. Its beneficial effect must be due to the relief of the distressing symptoms,—that is, by securing rest to the patient that cannot otherwise be obtained. It quiets the irritable heart, enables the patient to sleep, and thus his strength is somewhat restored and his circulation improved in consequence of the rest and of the increased excretion that take place during sleep.

Of the cardiac tonics the most valuable, doubtless, is *digitalis*. It acts on all kinds of muscle fibre, but especially on the heart, and next to that on the unstriated fibres of the arteries and the alimentary tract; least so on the skeletal muscles.

Its effect is to render the muscular fibre more elastic, so that it stretches out to a greater length and then contracts more nearly to its minimum length. This is illustrated in the heart when success follows its administration; the heart dilates more fully in diastole and contracts better in systole, both acts becoming more deliberate and effective.

Of the dosage there is a good deal of diversity of opinion. In slight disturbances to compensation, small doses may serve to steady the heart and restore its power, especially if complete rest is observed, but where, in chronic heart-disease, the failure is acute or marked, small doses have little effect. Our object can usually be attained only by what may be termed massive doses. It is common to meet with cases in which moderate doses do little if any good, but which are promptly relieved by large doses, such as the equivalent of three to five or even seven grains of the powdered leaf. Such doses are not dangerous if not repeated too frequently or persevered with too long. The effect of *digitalis* persists for a long time, it is not evanescent, therefore it should be given infrequently,—not oftener than two or at most three times in the twenty-four hours. Given thus in dropsy with scanty urine, it should increase the flow of urine, and as long as it does so it is doing the work for which it is given, and its use is free from danger. A lessening of the quantity of urine is a danger signal indicating exhaustion of an over-stimulated heart. As its effect on the heart, once produced, lasts for several days, it can, and should, be stopped for a day or two after three or four days of such free administration, and thus avoid the danger of its so-called “cumulative” action. Occasionally even small doses of *digitalis* stimulate the stomach and bowels so much that nausea, vomiting, and diarrhoea are produced. It is on account

of this irritative effect on the digestive tract that it has much oftener to be withdrawn than from any over-stimulation of the heart. Reference has already been made to the class of cases in which digitalis is powerless for good.

Next to digitalis, strophanthus is generally regarded as the most efficient cardiac tonic. My own experience with it has not been satisfactory, possibly owing to the inferior quality of the drug obtained. It is said to act more powerfully on the heart and less on the peripheral vessels than digitalis. Its action is more prompt in cases of emergency.

If there is high arterial tension, the nitrites, of which nitro-glycerin is most frequently given, do much to relieve a laboring heart by lowering the peripheral resistance. They often prove of great use in angina pectoris. Given three or four times a day the recurrent attacks are not infrequently prevented. Attacks of so-called cardiac asthma, which are in reality *angina sine dolore*, are often relieved completely by the nitrites. A few years ago a medical friend who had suffered for nearly a year from cardiac asthma was completely relieved for some months by nitro-glycerin. At first he took two minims of the one-per-cent. solution, but had to gradually increase the dose until at last he was taking eighteen minims. By that time the dyspnoea had ceased to be paroxysmal and became continuous on account of the dropsical effusions.

Strychnine is very useful, especially when the heart-failure is only moderate. Given subcutaneously it is more effective as the absorption from the stomach is poor.

Potassium iodide is of great use in those cases in which there are inflammatory changes in the peripheral vessels as well as in those having a syphilitic history. Only small doses are needed. It aids very greatly in the effectiveness of digitalis.

Calomel is highly thought of by many in cases with dropsy. From one to three grains should be given three times a day, or even every three hours, opium being added if necessary to moderate diarrhoea. It is an old remedy, given with digitalis; when combined with this and squill it formed the old pill triplex, or Guys's pill.

Graduated exercise has long been employed to strengthen a weak heart. Stokes was the first to use this means of treatment and point out its advantages. He even resorted to violent exercise, having, it is said, some patients take exercise by running behind

their own carriages. In recent years Oertel has been chiefly instrumental in directing attention to this method of treatment and to the restrictions necessary to be observed in its judicious use. The heart, like every other muscle, is strengthened by stimulation, provided the stimulation is well within its powers.

The strengthening of the heart depends upon hypertrophy of its walls, and this, in turn, upon an abundant blood-supply. If the coronary vessels are small or diseased the amount of blood conveyed to the tissues of the heart will not be sufficient to enable them to hypertrophy duly, and the strength of the wall of the heart will not be equal to violent strain. The degree of benefit resulting from exercise will therefore be in proportion to the freeness of the coronary circulation.

In resorting to exercise, the effect on the respiration and the circulation should be closely observed. No exercise should be engaged in that cannot be taken with regular, easy breathing and without holding the breath. The effect on the heart should be to reduce the frequency of its action and make the systole more deliberate and complete.

In addition to graduated exercise, especially in the form of resistance movements, Dr. Schott, of Nauheim, has strongly advocated the use of saline baths with or without free carbonic acid.

The Nauheim waters are rich in salines, especially in chlorides of sodium and calcium, and some of them contain large amounts of free carbonic acid gas. The temperature of the bath varies from 80° to 95° F. Almost if not quite equally effective baths may be artificially prepared by adding from five to eight or ten pounds of sodium chloride and half a pound of calcium chloride to a bath of thirty gallons at a temperature of about 90°. The first baths should be weaker and the temperature higher; the strength is gradually increased, and the temperature lowered to 85° or even less. The duration of immersion at first should be about eight or ten minutes, to be increased to fifteen or twenty minutes after some days.

By those who have had extended opportunities of observing the effect of baths, the area of præcordial dulness and the apex-beat are said to be retracted towards the median line, sometimes as much as two inches, usually from one-half to one inch. The right border of præcordial dulness is retracted often even more markedly than the left.

To impregnate the bath with carbonic acid gas, sodium bicarbonate and hydrochloric acid are used; from one-half pound to two or three pounds of sodium bicarbonate being required for each bath, according to the strength desired, and twenty-five per cent. of the amount of ordinary commercial hydrochloric acid. The bicarbonate is dissolved in the bath with the other salts, and then the hydrochloric acid slowly added about five minutes before the bath is used.

In the few cases in which I have used these baths, in only one has the result been quite satisfactory.

CASE III.—James P., aged sixty-five. An old soldier. He was much exposed a few years after leaving the army. For the last five years he has lived quietly, doing light gardening. He has had some dyspnoea for the last three years. In this time he has had two or three attacks of failing cardiac compensation. He came to the Toronto General Hospital in the summer of 1896 suffering from marked heart-failure. He was treated for a month with digitalis, strychnine, purgatives, etc., but without benefit. Morphine hypodermically gave only temporary relief. The saline baths were ordered, the immersion being at first ten minutes, and later twenty minutes. The first bath caused great dyspnoea on entering it, which slowly became less, and was only barely relieved by the time he left the bath. This distress was present in each of the first four or five baths, so that he took them reluctantly. He was a stout man with much dropsy, hence the outline of the heart could not be definitely made out. During these baths there was no apparent improvement in the pulse. After each bath he was rubbed dry and wrapped in a blanket, a hot bottle placed at his feet, and some stimulant given. With the continuance of the baths improvement gradually set in, until by the end of the third week the dropsy had disappeared, the breathing was easy, and he slept comfortably at night. He left the hospital a month later in a comfortable condition, the heart's action quite regular, appetite good, and he was able to sleep all night lying in bed. During the time of treatment by baths no medicines were given except laxatives as needed and strychnine. The result of this case was eminently satisfactory.

CASE IV.—Wm. E., aged sixty-six. A civil servant suffering from great cardiac dilatation, with extreme dyspnoea, insomnia, and dropsy. He had been treated with strophanthus, digitalis, caffeine,

etc., before I saw him, but without improvement. Morphine, one-fourth grain, at bedtime gave a good night's rest; it relieved breathing and steadied the heart. Magnesium sulphate was given every morning to produce a free liquid evacuation. After three days the morphine was stopped and codeine and caffeine, each two grains, and calomel, one grain, given instead, with chloralamide, twenty grains, at bedtime. Digitalis was also given. This gave good rest at night and made him sleep almost all day, so that the chloralamide was omitted and codeine reduced to one grain. In a few days he became nauseated, and both digitalis and codeine were stopped. His progress was satisfactory, and a general tonic was ordered. To maintain a stimulus to the circulation, desiccated suprarenal capsule, two grains three times a day, was given. His restoration was very satisfactory under the circumstances. He resumed work in October, and remained well until November, when failure again occurred and increased gradually. It required morphine, one-half grain, to secure rest and sleep. Not improving well, saline baths were resorted to, but with only moderate benefit. He improved slowly and was able to go out somewhat afterwards. A few months later he died suddenly while sitting on the side of his bed about to take breakfast.

The benefit from the baths in this case was slowly manifested; it is possible that as good progress would have been made without them. These two cases indicate fairly well the results I have had from bath treatment.

There is some doubt as to the restrictions to be placed on the bath treatment of cardiac disease. It is probably unsafe in cases in which it would be dangerous to quickly raise the blood-pressure. Hence baths should not be given in cases of aneurism and of advanced arterio-sclerosis.

By way of summing up: it is evident that the most successful management of the ever-varying phases of cardiac failure requires a knowledge of all methods of treatment and the application of any or a judicious combination of several, as best suited to each individual case.

As in all diseases, it is the patient rather than the disease that we have to consider. With too many people it is impossible to avail themselves of the most effective means for their restoration to health; in such we have to use the best available means, giving the patient every possible encouragement in his struggle with his afflic-

tion. A hopeful, confident mental state aids greatly in the treatment of all ailments as well as in restoring vigor of body where there is no disease.

Our work is not only to cure diseases, but also to make the most of damaged organs; and probably there is no class of diseases in which we can do more in prolonging life, even a useful life, than in cardiac affections. The difference between curing and prolonging an active useful life is, after all, only one of degree; the cured case has only a longer lease of life, and even that is not a certainty.

# THE TREATMENT OF BRONCHIAL INFECTION BY EMETICS.

CLINICAL LECTURE DELIVERED AT THE PITIÉ HOSPITAL.

BY PROFESSOR ALBERT ROBIN,

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GENTLEMEN,—The presence in our wards of two extremely interesting cases will enable me to lecture to-day on the treatment of bronchial infection.

Infection of our tissues can occur in different ways. There can be local and external infection; infection of natural cavities which are in communication with the outside; and internal general infection, or even internal local infection, to be quite accurate. Setting aside the other forms, I shall to-day refer only to cases of infection of natural cavities inaccessible to external antisepsis.

As regards the intestine, experience has shown us that internal antisepsis is a chimera. There is only one way of treating intestinal fermentation: to empty the intestine by a cathartic,—whence the advantage in typhoid fever of keeping up a moderate diarrhœa.

In the present lecture I should like to convince you that the best way to lessen bronchial fermentation is to empty the bronchial tubes by an emetic; in other words, to show you that as a therapeutic agent an emetic stands in the same relation to bronchial infection and fermentation as a cathartic does to intestinal fermentation and infection.

Emetics meet several of the indications that present themselves both in acute and in chronic bronchial infection. This class of remedies—especially ipecac—has a direct action on the initial pulmonary hyperæmia, as well as on the repletion and obstruction of the bronchial tubes by pus at a more advanced stage of the disorder.

Ipecac modifies the pulmonary circulation by its action on the

vasomotor system, even when it does not cause emesis. This fact is undeniable, and is sufficiently demonstrated in the treatment of hæmoptysis by ipecac. As a modifier of congestion ipecac is therefore indicated in the early stages of bronchopneumonia.

It also meets another indication,—that of mechanically emptying the air-passages. In this connection it acts in two distinct ways: by stimulating bronchial secretion, and by causing emesis. The secretion it brings on consists of fluid mucus, which has a tendency to liquefy the muco-pus that has thickened by stagnation, and will facilitate its elimination; the emetic also favors spontaneous propulsion of the accumulated secretion by stimulating the contractile energy of the Reissessen fibres. Finally, the effort and violent muscular contraction connected with emesis produces immediate evacuation of all the air-passages,—a mechanical sweeping out or emptying of the tubes, as it were.

This is the relief that can be afforded for bronchial infection by the administration of an emetic, but some one may reply, Cannot the same result be accomplished in a less violent and disagreeable way, as the production of emesis is always unpleasant?

You are all aware, no doubt, that creosote has been extensively used for this purpose, as it favors bronchial secretion and is believed to be a bronchial antiseptic. In this way it has been used on an immense scale in tuberculosis.

I am obliged to say that the hopes that had been based on the action of creosote in this connection have practically fallen to the ground. In fact, Renaut states that creosote has done more harm to consumptives than Koch's tuberculin! In trying to disinfect the surface of the bronchial mucous membrane by the administration of creosote we do harm to the digestive process, beyond question, and tubercular patients depend for their recovery almost entirely on satisfactory digestion. Furthermore, the amount of creosote that is set free on this mucous surface is, as a matter of fact, quite infinitesimal. Creosote can and has been given in inhalations, vaporizations, etc., but there is serious doubt whether by this method the active substance ever reaches the diseased parts of the lungs.

For these reasons there is no comparison to be made between the action of the class of substances of which creosote can be taken as an example, and the emetics of which ipecac is a type, in the treatment of bronchial infection. Laënnec, not to speak of Hippocrates,



was an advocate of the emetic in such cases, and he cites the case of a lady aged eighty-five who had suffered from chronic bronchial catarrh with profuse expectoration for eighteen months, and to whom he gave an emetic fifteen times in the space of one month. She was living eight years later!

The best emetic for adults is ipecac; for old people, ipecac with tartar emetic. I usually give one and one-half grammes, or twenty-three grains, of ipecac with five centigrammes, or three-fourths of one grain, of tartar emetic, to be divided into three powders, one of which should be taken at intervals of a quarter of an hour in half a glass of water; if one powder, or two powders, have a sufficient action the entire dose need not be taken. As soon as vomiting begins the patient should drink tepid water, so that the stomach shall have something upon which to contract.

It is scarcely necessary for me to tell you that emetics are commonly used in the disorders of childhood, where their efficacy, proved empirically, has made them one of the leading agents of popular medicine.

You have on a number of occasions been able to verify in our wards the advantages connected with the use of emetics in cases of bronchial infection so common among old people, and we have at present under treatment two such patients, whose clinical history I shall now briefly relate.

The first case is that of a man aged sixty-three, who came to us suffering from violent dyspnœa, which was rendered still more unendurable by pain in the left side of his thorax. There was abundant, purulent expectoration; the temperature fluctuated in the neighborhood of 39° C., or 102.2° F., going down a few tenths of a degree in the morning, and the tongue was dry and thickly coated. The base of the left lung was dull on percussion; auscultation in that region showed a hard but limited bronchial souffle, and a focus of fine, subcrepitant râles. The patient was emphysematous, and there were disseminated bronchial râles all over the rest of the chest. There was no cardiac lesion, and the urine was scanty and slightly albuminous. Bacteriological examination of the sputa showed streptococci in abundance.

We made the diagnosis of bronchopneumonia, which was confirmed by the course taken by the attack.

On the following day, in spite of the application of a blister to

the base of the left lung and the administration of antimony as an expectorant, the temperature rose to  $40^{\circ}$  C., or  $104^{\circ}$  F., the expectoration was troublesome and thick, the dyspnœa was violent, and there was no modification in the physical signs. In face of such a situation I had no hesitation in prescribing an emetic, and he was given one and one-half grammes, or twenty-three grains, of ipecac with five centigrammes, or three-fourths of a grain, of tartar emetic, in divided doses.

Let me draw your attention specially to the patient's time of life. He is sixty-three years old, and it would seem that this case was an unfavorable one for demonstration. This, however, is a mistaken opinion. Bronchopneumonia occurring in old people can be very advantageously treated by emetics, and you must not be deterred from their use by the generally accepted apprehension of causing collapses or cerebral hemorrhage. While I was physician to the Hospice des Ménages, devoted as you know to the exclusive treatment of very aged people, I used emetics on a large scale with the most satisfactory results.

The patient's temperature, which, on the evening before the administration of the emetic, was still  $39.6^{\circ}$  C., or  $103.2^{\circ}$  F., fell to  $38.3^{\circ}$  C., or  $100.9^{\circ}$  F., the following morning, and then steadily returned to normal. Dyspnœa decreased, the expectoration became more fluid (filling two cups a day), and in three days the attack of bronchopneumonia was relieved. Auscultation then showed that the souffle had disappeared, leaving only a few loose râles.

Six days later a new outbreak appeared and his temperature again began to rise; but another emetic was given, which reduced his temperature to normal in three days.

Finally, with the use of expectorating remedies and dry cups from time to time, this man, who was in advanced years and whose constitution had been severely shaken by serious bronchial and pulmonary infection, was restored to a satisfactory condition of health after a period of convalescence lasting three months.

With this instance can be compared another *acute* case of bronchial infection treated by emetics, though in a much younger man. The patient, thirty-five years of age, had suffered for several days from bronchopneumonia caused by influenza. Temperature about  $40^{\circ}$  C., or  $104^{\circ}$  F.; chest was full of mucous râles; intense dyspnœa; marked cyanosis, and pulse thready and feeble, so that a fatal issue seemed imminent.

In spite of his serious condition I administered an emetic. That very evening there was marked improvement, breathing being freer, pulse better, and the patient feeling relieved. The temperature had fallen one degree, and reached the normal by the following evening; a few days later he left the hospital cured.

These two were cases of *acute* bronchial infection cured by emetics, but their action is equally satisfactory in *chronic* cases, which you will generally meet with in persons well advanced in life.

The other patient of whom I spoke as being in our wards is a type of this chronic intoxication. He is fifty-six years of age, a day laborer, without hereditary pathological antecedents. In his previous history we find a reference to two attacks of general acute articular rheumatism, the first at the age of twenty and the second twelve years ago. He has had frequent coughing spells for twenty years past, particularly during the winter season. He has noticed for some time past that he gets out of breath easily, especially on going up-stairs. During the past two months his cough has increased, as he caught a fresh cold, and his expectoration is extremely abundant. His general condition is satisfactory; in physique he is robust and vigorous.

Auscultation gives the usual signs of pulmonary emphysema combined with chronic bronchitis. There are whistling, sonorous, mucous râles all over the lungs. At the left apex posteriorly percussion shows slight dulness, and the ear applied to the same point reveals a focus of moist, subcrepitant râles.

The heart is enlarged, the point striking in the sixth interspace; the sounds are muffled, but there are no murmurs. Heart-action somewhat irregular. Arteries hard and encrusted.

The other organs show nothing of special interest. Urine is abundant and contains traces of albumen and urobilin, a sign of hepatic insufficiency.

In a word, he is an old rheumatic patient with arterio-sclerosis, pulmonary emphysema and chronic bronchitis with, possibly, bronchial dilatation, as the daily quantity of mucopurulent expectoration is very considerable.

But our patient's temperature is above the normal, and emphysema and bronchial dilatation do not cause fever. We have, therefore, a certain degree of bronchial infection. The temperature, which was 38.4° C., or 101.1° F., when he entered our wards, rose to 39° C., or 102.2° F., the days following, where it remained. In

addition to this the patient had anorexia, slept badly, and lost his appetite and some weight,—two kilos, or four and four-tenths pounds, in ten days. The local stethoscopic signs at the left apex became more marked; dulness became complete, and the râles more moist and audible over a wider area.

In view of the bad general condition, fever, and increase in the stethoscopic signs at the left apex, the first idea that occurred was that of tubercular breaking down of the lung; but careful bacteriological examination of the sputa, made a number of times on different days, failed to detect tubercle bacilli.

Examination of the respiratory chemistry showed decreased interchange, as is customary in pulmonary emphysema, and not an increase, as is usual in pulmonary tuberculosis.

An emetic brought the temperature down from 39.4° C., or 102.9° F., to 37.3° C., or 99.1° F., and a second one, given two days later, reduced it from 38.6° C., or 101.4° F., to 37.9° C., or 100.2° F. The patient then felt much relieved; his cough was less distressing, dyspnœa had disappeared, and his night's sleep was satisfactory. The râles at the left apex had decreased, and the urine only showed traces of albumen. The analysis of the respiratory chemistry showed that interchange had increased under the influence of the emetic; the patient absorbed twice as much air per kilo of weight after the emetic, and consequently twice as much oxygen. He also eliminated twice as much carbonic acid per kilo per minute, and these results of the emetic persisted for twenty-four hours after its administration.

Encouraged by this improvement and as the bronchial obstruction was disappearing, I no longer gave the ipecac in emetic doses, but in very small doses. To obtain its action as an expectorant and as an agent for moderating congestion, I prescribed the following preparation:

R White oxide of antimony, 1 gramme; or grs. xv;<sup>1</sup>  
 Tincture of *fresh* aconite root, 20 drops; or gtt. xx;  
 Tincture of belladonna, 10 drops; or  
 Tincture of nux vomica, 10 drops; or aa gtt. x;  
 Syrup of ipecac, 15 grammes; or  
 Syrup of opium, 15 grammes; or aa ℥iv;  
 Simple syrup, q. s. ad. 150 grammes; or ℥v.  
 Sig.—Tablespoonful every hour until vomiting occurs.

<sup>1</sup> In the United States the white oxide of antimony is seldom employed because of its insolubility unless an acid be present. Preference is given to the tartrate of antimony and potassium, the emetic dose of which for an adult is one grain.—J. D.

In this compound formula the antimony is an expectorant; the belladonna is given to dry up the mucous surface; the aconite to dry the surface and to lessen the bronchial reflexes; the nux vomica to stimulate the Reissessen fibres and, by bringing them into action, to lessen the bronchial dilatation.

The amount of ipecac given was, however, too great, and in a few days the patient began to feel nauseated; but this condition ceased when we had reduced the syrup of ipecac to five grammes, or seventy-five grains, in our formula.

Thanks to the use of the emetics the temperature returned to normal, and since then has not risen above  $37.5^{\circ}$  C., or  $99.5^{\circ}$  F. The râles at the left apex gradually disappeared, and three weeks after the treatment was commenced the patient left the hospital completely cured.

Another case somewhat resembling the preceding was one of asthma and bronchitis, and here again the emetic was efficacious, the dyspnoea becoming less distressing, the expectoration decreasing, and the attacks of asthma growing less frequent. In this case also the analysis of the respiratory chemistry, made before and after the administration of the emetic, gave us most interesting and instructive information, showing that respiratory interchange was energetically stimulated by the use of this therapeutic agent.

Pulmonary ventilation increased nearly twofold. The oxygen used by the tissues per kilo, or cubic inch, per minute went up from 0.569 cubic centimetre, or 0.224 cubic inch, to 1.500 cubic centimetres, or 0.590 cubic inch, while the carbonic acid eliminated per kilo and per minute rose from 2.137 cubic centimetres, or 0.841 cubic inch, to 5.1006 cubic centimetres, or 2.000 cubic inches.

These examples will suffice, although I could give you many more, to prove the powerful action of emetics on pulmonary ventilation and capacity, on the formation of carbonic acid, and on the amount of oxygen consumed, which are all increased in large proportions.

This increase in gaseous interchange of all sorts effected by the use of emetics consists in two mechanical factors, increased respiratory capacity and ventilation, and in one chemical or vital factor, greater absorption of oxygen and formation of carbonic acid per volume of air used. In view of this twofold mechanical and chemical effect of emetics, we have reason for placing our faith, as did

the physicians of ancient times, in their efficacy, at any rate as applied to bronchial infection.

I have no hesitation in saying that for this purpose they are infinitely more valuable than any antiseptic. They empty the bronchial tubes, which is the best form of antiseptics, and in addition to that they are powerful aids to oxidation, which is one of the most efficacious means we possess to free ourselves from microbe toxins. There is no antiseptic at present known that can equal them in effect.

# THE TREATMENT OF FUNCTIONAL AND LATERAL CURVATURE.

READ BEFORE THE ORTHOPÆDIC SECTION OF THE NEW YORK ACADEMY OF  
MEDICINE.

BY JAMES K. YOUNG, M.D.,

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## THE TREATMENT OF FUNCTIONAL AND LATERAL CURVATURE BY LIGHT GYMNASTIC EXERCISES.<sup>1</sup>

MY attention was first attracted to this subject about nine years ago, by Dr. John M. Keating, of Philadelphia, and Miss Johnson, a Swedish masseuse, who had treated several cases successfully by the use of light gymnastic movements adapted from the Swedish system; some of which I was associated with, and one of these furnished the illustrations of the eight primary movements in the article on "Physical Development." In this case the patient was entirely cured, has remained permanently restored, and has ever since been teaching gymnastics.

Since that time I have devoted considerable thought and study to the subject, and have completed a system by which a large number of these cases may be permanently cured. This form of treatment is particularly adapted to the treatment of the mild and medium degrees of the atonic or habitual variety of the affection. This system consists of four parts:

*First.* Development of weak muscles by exercises adapted to them individually or collectively.

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<sup>1</sup> Read before the New York Academy of Medicine, Orthopædic Section, November 20, 1896. Keating's Cyclopædia of Diseases of Children, vol. iv.

*Second.* Slight over-development of the weak muscles.

*Third.* Uniform development of all the muscles.

*Fourth.* The use of eight special movements to prevent relapse.

Dividing the spine into its three regions, cervical, dorsal, and lumbar, we may have two curves in each, as the spine is deflected to the right or left. The entire spine may be deflected to the right or left, making in all eight primary curves:

1. Right cervical primary scoliosis.
2. Left cervical primary scoliosis.
3. Right dorsal primary scoliosis.
4. Left dorsal primary scoliosis.
5. Right lumbar primary scoliosis.
6. Left lumbar primary scoliosis.
7. Right total scoliosis.
8. Left total scoliosis.

In my observation the greater number of primary curves are of the right dorsal variety, the left lumbar being next in order of frequency, and the left dorsal being third. The others occur with greater rarity. The three degrees that are recognized, the mild, the medium, and severe, are determined by the self-suspension of the patient, the prone position, or with slight pressure upon the curve with the hands. If the curve is entirely overcome by self-suspension, or prone position, or manual pressure, it is of the mild degree. If the curve is influenced by self-suspension, prone position, or manual pressure, but not entirely straightened, it is of the medium degree. If the curve is not influenced by self-suspension, or these other means, it is of the severe degree.

As in other deformities, primary forms of curvature are exceedingly rare. If the primary curve exists, sooner or later a secondary or compensatory curve will be established by the efforts of the spinal muscles to re-establish the equilibrium. If the functional curve persists long enough a third or tertiary curve will be established. Thus, a primary curve in the left lumbar region may lead to a secondary curve in the right dorsal region, and ultimately to a tertiary curve in the left cervical region; or a primary right dorsal curve may lead to a secondary left cervical curve, and a tertiary right lumbar curve. The production of these functional curves is exceedingly interesting; most of them are produced by faulty positions assumed through carelessness, fatigue, or occupation. The ex-

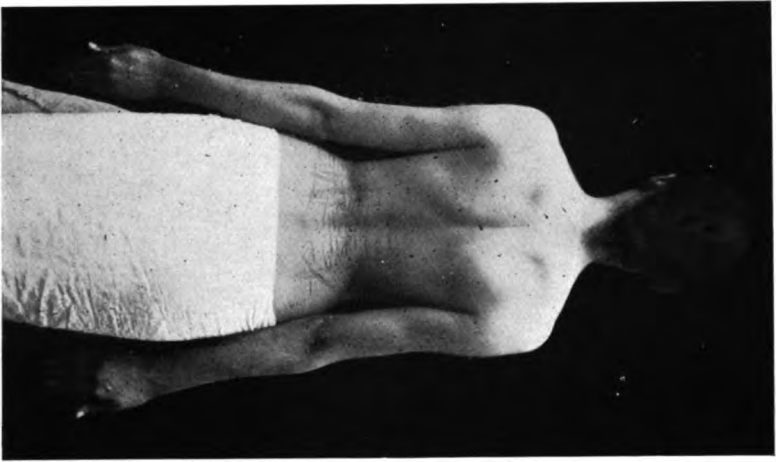


planation for the greater relative frequency of right dorsal and left lumbar curves is due to the faulty habit of young individuals resting when fatigued upon the left limb, the pelvis being lowered on the left side, and the left knee bent slightly forward. This curves the spine in the lumbar region, with the convexity to the left, and in the dorsal region with the convexity to the right. This explanation is well illustrated in a young artist's model, by a rapid production of a left dorsal curve, from standing for long periods upon the right leg. These faulty positions lead to an elongation of the bent and relaxed limb, and a softening and loss of tone of all the muscles of the relaxed lower extremity; so that when the individual assumes the best standing position, the relaxed limb is apparently longer than the normal limb, upon which the weight has been borne.

The plan of treatment consists in detail of finding the best voluntary position that can be assumed by the patient to correct the deformity, and then taking all the exercises in that position, or favoring that position in the use of all the movements. This leads to a development of the relaxed or contracted muscles on the concave side of the deformity, and the stretched and atrophied muscles on the convex side; and these exercises are continued or modified until there is a slight over-correction of the deformity. The general physical development is then commenced, and continued until a cure is accomplished.

Each case is an individual study, and as primary forms are exceedingly rare, and while individual sets of exercises are prescribed for individual curves, most formulæ for exercises will consist of combinations of sets of exercises. These exercises must be taken under the personal direction of the surgeon, or a trained instructor or masseuse. They must be taken slowly once a day, from five to fifteen times each, and two hours after, or one hour before a meal. The exercises are followed by massage of the weaker muscles; and the patient must lie in the prone position, or key-note position, for half an hour to an hour after. This latter position will differ in each variety of curve. The key-note position for right dorsal scoliosis, the most common form, is with the right arm extended sideways, and the left arm elevated by the side of the head. The key-note position for left dorsal scoliosis will be the reverse of this.

Medical treatment is to be prescribed if required, and any menstrual irregularity corrected. The exercises are continued daily for



**FIG. 1.—No. 1 Exercise. Best standing position, head up; chin in; chest up and forward; weight on balls of feet.**



**FIG. 2.—No. 2 Exercise. Key-note position, which is, left arm upward, stretch; right arm sideways, stretch.**

matic  
bar  
wh  
led  
sp  
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wel  
lef  
g.  
la  
e  
e  
e  
e



FIG. 8.—No. 4 Exercise. Same as No. 2, with "trunk to right bend."



FIG. 4.—No. 8 Exercise. Key-note, right foot forward, fall out.





FIG. 3.—No. 4 Exercise. Same as No. 2, with "trunk to right bend."



FIG. 4.—No. 8 Exercise. Key-note, right foot forward, fall out.



from four to six months, excepting Sundays and during the menstrual periods. The exercises are given three or four at a time by the surgeon, until the whole number has been learned. They should occupy from twenty to thirty minutes, and should never be carried to the point of fatigue. Throughout the exercises breathing should be regularly carried on, and at the end of each exercise one or two full breaths should be taken. In all these exercises the most important is time; from four to six months will be required to cure mild cases, and two to three courses of the same length will be required for medium cases. The exercises are usually given under the personal observation and charge of a masseur or a masseuse, the surgeon directing the treatment from week to week, or more frequently if the case demands; it being absolutely necessary that the patient strictly complies with the method of treatment directed by the surgeon. To obtain the best results where the school hours conflict, it is necessary to abolish them altogether during the entire course of treatment; patients must devote themselves conscientiously to the means prescribed for their recovery, and not be distracted or interfered with by social functions.

The method can best be illustrated by considering the special treatment of primary right dorsal scoliosis. In right dorsal scoliosis the right shoulder is elevated, with the scapula projecting backward, the left shoulder depressed, the scapula flattened, and the spinous processes of the dorsal vertebræ rotated to the right, the relaxed and contracted muscles being on the concave side, the stretched, thinned, and fatty degenerated muscles on the convex side of the curve. The pelvis is tilted downward to the left, the right leg is elongated, and the spinous processes of the lumbar vertebræ are rotated to the left, and the left hip projects a little forward. The treatment is directed to the muscles of the curve, by placing the skeleton in such positions that the muscular attachments are more normal, and can therefore be contracted and strengthened. In addition to this, the right or long limb is shortened by special exercises, which contract the muscles about the hip-joint.

That the long limb can be shortened is a well-established fact, illustrated in the cured cases; and especially in a case of injury to a college student under my personal observation, in which elongation had occurred from relaxation following the injury. The history of the case is as follows: H. B. W., aged eighteen years, consulted



me June 20, 1894, suffering from injury to the right knee and hip received five weeks before, from being run into by another player during a game of base-ball. Forced flexion was greatly limited, the adductors were wasted; the pain was a localized soreness about the great trochanter. The following measurements were taken: Right limb,  $36\frac{1}{2}$ ; circumference, at 7 =  $18\frac{1}{4}$ , at 12 =  $21\frac{1}{4}$ . Left limb, 36; circumference, at 7 =  $19\frac{3}{4}$ , at 12 =  $21\frac{1}{4}$ . Right calf,  $14\frac{1}{4}$ . Left calf,  $14\frac{1}{4}$ . These showing a lengthening of the right limb of three-quarters of an inch. For a time he was treated by extension to the right limb, with massage. Passive movements were gradually added, and the limb shortened in length until they became equalized, and the muscles becoming firmer and harder in the affected limb.

This is shown in three other cases which have come under my observation, and in which injury to the hip was followed by lengthening of the limb. These cases were corrected by appropriate treatment. The first case was that of a girl who had injured her hip by fancy dancing. The second case was of a girl of four years of age who injured her hip and knee by falling against a trunk. The third case was a girl of ten years, where injury to the hip was the result of slipping down three steps; she missed two and struck the third. Upon recovery of the hip injury, after three months' treatment, it was found that the lengthening was half an inch, and the muscles were softened and relaxed.

In cases where the inequality of the lower extremities is due to any other causes, as flat-foot, asymmetry, or fracture, these should be corrected by adding to the sole of the shoe worn on the foot of the short limb, or other proper mechanical appliances. The above remarks, however, apply particularly to the elongation occurring in atonic cases. That elongation and rotary lateral curvature may occur from faulty postures long continued is well exhibited in the case of the professional artist's model before referred to, who within three months developed a left dorsal scoliosis from standing on the right limb.

The special exercises in right dorsal scoliosis are as follows:

1. Best standing position, head up; chin in; chest *up* and *forward*; weight on balls of feet.
2. Key-note position, which is, left arm upward, stretch. Right arm sideways, stretch.



FIG. 6.—No. 9 Exercise. Key-note, hanging, left hand higher, contraction of left limb with resistance.



FIG. 6.—No. 7 Exercise. Key-note, same as No. 6, "heels raise" with "knees bend."





FIG. 7.—No. 5 Exercise. Key-note, "trunk diagonally forward, bend."



3. Same as No. 2, with "trunk forward, bend."
4. Same as No. 2, with "trunk to right, bend."
5. Same as No. 2, with "trunk diagonally forward, bend."
6. Same as No. 2, with "heels, raise."
7. Same as No. 6, with "knees, bend."
8. Same as No. 2, with "right foot outward, fall out."
9. Hanging, left hand higher, contraction of left hip with resistance.
10. Hanging, right or left leg sideways raise with resistance.
11. Hanging, right or left leg sideways raise, and downward sink with resistance.
12. Lying supine, "right knee upward, bend."
13. Lying supine, start with "right knee upward, bend," and then "right knee downward, stretch."
14. Lying supine, right hip rotation, starting up, and also starting outward.
15. Lying prone, take position as in No. 2, "trunk backward, bend, and trunk to the right, twist."
16. Salute,—first count, right arm forward, bend; on second count, "right foot outward, fall out;" third count, "right arm sideways, fling;" fourth count, standing position.
17. Left shoulder elevated, right scapula depressed; passive movement.

These include exercises for the right lumbar secondary curve, which is usually present in these cases.

The exercises for the treatment of left dorsal scoliosis are exactly the reverse of these.

The exercises for lumbar scoliosis, right or left, include all the exercises in the foregoing for the development of the lower extremities, with exercises added for the special treatment of the lumbar muscles. These are principally leg exercises given in the hanging position, or resting the weight of the body on the limb on the side of the concavity.

In the treatment of primary cervical curves, after the removal of the cause, special exercises with resistance are recommended to develop the muscles of both the concave and convex side of the curve, and when associated with right dorsal scoliosis they are directed to the right side of the neck, the head being carried well to the left, and when associated with left dorsal scoliosis these exercises

are in the reverse of this Hanging movements are also very useful.

After the curves have been over-corrected by these methods general exercises are employed, with or without the use of the apparatus, for the symmetrical development of the body. The positions assumed in these exercises are with arms extended, arms elevated, and neck-rest position. After this has been accomplished, the general muscular development is maintained by the eight special exercises originated by Dr. Keating and myself seven years ago, and published at that time.<sup>1</sup> They are as follows:

1. "Neck firm." Then "heels, raise."
2. "Neck firm, trunk to left and right twist."
3. "Arms upward, stretch." Then "trunk to left and right, bend."
4. "Arms upward, stretch." Then "trunk forward, bend."
5. "Right (or left) arm upward, stretch." Then "right (or left) foot outward, fall out."
6. "Arms upward, stretch." Then "right (or left) foot outward, fall out." Then "trunk twist towards side corresponding to forward foot."
7. "Arms sideways, raise." "Right (or left) foot forward, fall out."
8. "Arms upward, stretch." "Heels, raise." "Knees, bend."

If relapse should occur, after using the exercises with arms extended, arms elevated, and the neck-rest position, the patient should return to the key-note position until the curve is again slightly over-corrected. While the patient is taking these exercises with the arms extended and elevated, and in the neck-rest position, light dumb-bells may be employed with great advantage in the development of the general physical condition.

It is most particularly important that children should be examined early in life, and if there be any symptoms of curvature, they should be treated before they have advanced beyond a degree which will not admit of perfect recovery.

#### Conclusions:

*First.* Mild and medium cases of scoliosis can be cured by light gymnastic exercises.

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<sup>1</sup> Keating's Cyclopædia of Diseases of Children, vol. iv.



FIG. 8.—No. 10 Exercise. Key-note, hanging, right or left leg sideways, raise with resistance.



FIG. 9.—No. 15 Exercise. Key-note, lying prone, take position as in No. 2, 'trunk backward, bend, and trunk to the right, twist.'







**Fig. 10.—No. 16 Exercise. Salute.**



**Fig. 11.—No. 17 Exercise. Key-note, left shoulder elevated, right scapula depressed; passive movement.**



*Second.* The exercises are directed to the development of the weaker muscles.

*Third.* The exercises should slightly over-develop the weaker muscles.

*Fourth.* General exercises for the uniform development of all the muscles.

*Fifth.* The general physical development should be maintained by the use of the author's special exercises.

[NOTE.—The illustrations are from photographs of a "model." When the patients take the exercises, they wear a special gown which only exposes the back.]

## TREATMENT OF ACUTE INFLAMMATORY MIDDLE-EAR DISEASE.

BY J. HERBERT CLAIBORNE, JR., M.D.,

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It is not my purpose in this article to make a lengthy *résumé* of all the methods employed in the treatment of acute middle-ear disease. It is my purpose to state in as plain and succinct a manner as possible those methods which have met with success in my own hands.

In treating acute otitis media it is well to remember that the region involved is one very much circumscribed on account of its anatomical characteristics. Towards the external world lies the drum; towards the brain lies the bony wall on which may be seen the promontory and the round and oval windows; in the rear is the opening into the mastoid cells or antrum; to the front is the opening of the Eustachian canal. It is not to be forgotten that this chamber is traversed by a chain of ossicles, one of which is attached to the ear membrane and another fits over the oval window. Inasmuch as inflammation in any region must necessarily produce products which may be termed foreign, it stands to reason that this circumscribed area must be in a measure filled under these circumstances. Now, in the natural condition of things the drum should move inward and outward according to the vibrations of the atmosphere against it. If this chamber is filled with the products of exudation, or the lining membrane of this chamber be swollen, it must necessarily follow that the drum will not be able to make its normal physiological excursions, and, since the phenomenon of normal hearing is dependent directly upon the proper oscillations of the drum, it follows that the absence of physiological hearing occurs. In plain terms, the patient must be more or less deaf.

Owing to the extreme delicacy of all the structures involved,

the near presence of the brain, and the necessary distention of the drum when an exudate exists, pain, more or less severe, must always attend inflammation in this region. Pain and deafness, then, are the two most prominent symptoms to be found in *otitis media catarrhalis acuta*. Another symptom which is quite prominent is the thumping which is complained of so bitterly by those suffering from this affection. This in a large majority is due to the surging of the blood in the carotid canal. If headache be taken into consideration, we find we have enumerated the main symptoms of this affection.

Now, there are several forms of acute inflammation of the middle ear, but the form to which I call your particular attention is that which is known as acute catarrhal middle-ear inflammation, by which I mean an inflammation in which serum is poured out in this cavity. Of course, an acute earache may be ushered in by the presence of pus when the surgeon sees the case, but in the nature of things a catarrhal serous inflammation must precede this.

It seems to me that the therapy we have in these cases is outlined in the brief description which I have given of the pathological condition. The object primarily is to get rid of exudation when it exists, or to alleviate and remove the swollen condition of the membrane when this condition alone exists. For that purpose the treatment which is to be employed must be constitutional and local. Of course, this disease occurs in the adult and in the child. This article is written equally for both of them, but it is well known that the disease occurs more frequently in childhood than in adult life.

The appearance of the drum as revealed by the otoscope differs with the severity of the attack. The drum may simply appear to be red. In mild cases the periphery of the drum, that portion which is along the hammer, is red. In severe cases the entire drum is red and swollen, and frequently blood-vessels may be seen coursing from the periphery to the handle of the hammer. In severer cases, when there is a large amount of exudate in the middle ear, the drum may be bulged outward and give the appearance of a half-ball or even that of a polypus. Again, when the amount of serum is considerable and the drum itself is not very much swollen, careful inspection may reveal a line which marks the limit of the exudate. This line is apt to be curved in the form of a crescent. I lay particular stress upon these appearances of the drum because when

they exist the treatment must be modified in a measure from that which is usual.

The most frequent cause of this disease is the "catching of cold," whatever that may be, which is attended, as a rule, by an acute inflammation of the pharynx, the post-rhinal region, and the nares. In the summer-time bathing is the most frequent cause. Those of us who work in the large clinics of a city situated upon the water are accustomed to see every summer many cases of acute catarrhal inflammation of the middle ear in boys who go swimming.

The cases likely to give the surgeon the most trouble and those to which it is my purpose to apply these words more particularly are the cases which occur in extremely small children and which are apt to come on suddenly, most frequently in the middle of the night. The surgeon is called to see the child, who awakens in the middle of the night screaming with pain. The natural application of the child's hand to the sore spot indicates to the parent what the trouble is. On examination, the surgeon is apt to find a drum with some of the characteristics which have been outlined, and he is called upon to relieve the suffering of the little patient as soon as possible. I consider these cases most difficult to treat. After the diagnosis has been made, my invariable rule is to instil into the ear as soon as possible a hot four-per-cent. solution of cocaine. I have never seen any untoward effects from this, and it has been my fortune to see in a very few minutes a subsidence of pain. I am accustomed to prescribe immediately thereafter an anodyne such as is fit for children,—usually paregoric or tincture of opium in proportion to suit the age of the patient. Unless the child goes to sleep at once, I then order a hot solution of boracic acid to be dropped into the ear every ten or fifteen minutes until the pain has subsided still more or the anodyne has commenced to have its soothing effect. If the patient goes to sleep, I leave the case until morning, when I am accustomed to prescribe a very simple remedy, and that is a saline purgative in a good big dose. If the child is under twelve years of age, I find a tablespoonful of Epsom salts the most efficient remedy that I have at my command. I am not afraid to give that much Epsom salts to children younger than that, for I have never seen any ill effect come from it when used with anything like reason. It is well known that the Epsom salts is a saline purge, and that it is also a hydrogogue purge, and that it causes the exuda-

tion of a large amount of water through the blood-vessel walls into the intestine, and that this is carried off through the natural ways. I hold there is no more certain way of getting the water out of a bucket than by knocking its bottom out, and when I prescribe Epsom salts in proper dose in a person suffering from this disease, I feel there will be an extraction of water from every part of the system, and it has been my experience that the relief of the inflammation of the middle ear is rapid, certain, and safe.

In treating a case of otitis media in an adult, I of course first inspect the drum, and if the condition of affairs does not warrant immediate surgical interference, I prescribe a full ounce of Epsom salts before any other measure is employed. When the drum is swollen in the manner I have indicated, so as to exhibit a bulging appearance, I consider the classical thing to do is to perform paracentesis, but my experience teaches me that if one will prescribe Epsom salts and relieve the pain by the instillation of cocaine and hot boracic acid, paracentesis of the drum will not be necessary as a rule. If, however, this is to be done, I favor the method of incision which is practised by Schwartz and recommended by von Troeltsch, which is to say, a distinct linear incision in the posterior inferior quadrant of the drum, and I wish to lay stress upon the fact that an *incision* is made and not a puncture. When this has been accomplished, it will be found that there will be a subsidence if not entire relief of the pain and swelling. If sufficient relief is not obtained, a further procedure should be employed. Having cocaine-ized the nose thoroughly, the Eustachian catheter should be introduced and a column of air be gently blown through it into the ear from a hand-bulb, which ought to be attached to the coat of the operator after the manner employed by Schwartz. I wish to enter my protest against the introduction of the beak of the Politzer bag into the mouth of the catheter as a means of inflating the middle ear. There are few surgeons whose hands are steady enough to compress the bulb sufficiently to throw air into the middle ear without at the same time injuring to some degree the membrane in or near the Eustachian tube. In children I think we ought to employ every means possible before we make use of the surgical means of relieving pain. Nothing frightens a child who is ill so much as the notion of having an operation performed upon it. I have seen a child suffering agony from acute middle-ear inflammation thrown



into a spasm simply by a demonstration of the instruments and an attempt on the part of the surgeon to incise the drum. I am willing to incur the condemnation of those who are scientific at the expense of humanity by saying that I believe great harm will be done under such circumstances, and that it is better to trust to spontaneous rupture of the drum. In plain words, it is my custom not to incise the drum of little children, but to rely upon the treatment which I have outlined and, I trust, explicitly stated.

I remember two particular cases of otitis media occurring in adults in which the use of Epsom salts produced an effect which was almost as rapid, and certainly as satisfactory, as could have been obtained by surgical measures. A young man of about twenty-five called me up in the middle of the night on account of an acute ear-ache. The drum presented the characteristic appearance of acute inflammation of the middle ear. It was swollen and slightly bulging. I cocainized his nose thoroughly and used the Eustachian catheter to my own satisfaction, and then prescribed one ounce of Epsom salts to be taken immediately. He went to a neighboring drug-store, took the Epsom salts, and sat down to wait. In due time he had three or four profuse, watery evacuations, with an almost total subsidence of pain. It was present in this case and I have observed it in others, that as soon as the Epsom salts commenced to pass into the general system the pain began to diminish, and that the greatest amount of relief was experienced when the salts had produced their classical physiological result.

I have on my list a robust physician of thirty-eight years of age who was accustomed to have during the summer acute otitis media. On the first occasion of my seeing him the drum was distinctly swollen and bulging, and I was much tempted to make paracentesis. However, he expressed such a disinclination to have any instruments used upon him that I decided to rely on the Epsom salts, and ordered him to take an ounce, no more and no less, at once. In a few hours he returned to say that the effect had been everything that could be desired. His pain was entirely relieved, his hearing partly restored, and the drum was very much less swollen and bulging. The application of the Eustachian catheter brought the engagement to a finish, and subsequent treatment of his nasopharynx for a day or two restored him to his normal condition.

This physician has repeatedly come to me with this trouble, and in every instance I have relieved him in the way I have described.

It is taught by Schwartze and others that when the acute symptoms of tenderness have disappeared and in certain cases, particularly in those in which the exudate in the middle ear is tough and slimy, syringing of the middle ear through the Eustachian catheter and through the rent in the drum is a proper and efficient method of treatment. I had the good fortune to count Schwartze among my masters, and I have seen this method of treatment employed with great success in his clinic at Halle. I make an observation which will come home to all, I believe, when I say that German peasants are not Americans, and, while in the hands of a skilful, clever manipulator this method of treatment is possible and may bring success, I believe that the majority of those who suffer would object to its employment, and I cannot say that I am convinced it has any peculiar advantages over simpler methods of treatment. In short, I no longer employ it.

The subsequent treatment of this affection is within the hands, one might say, of almost any one who has medical knowledge. It consists in attention to hygiene of the nose and throat and, possibly, the application of nitrate of silver to the nasopharynx. The mild employment of the catheter or the Politzer bag is also indicated. It is my purpose here to dwell upon the treatment of the acute symptoms. I cannot force myself from the conviction that in the case of little children of very tender years an anodyne for the pain is the main thing which we rely upon when called to a case in the agony of an acute attack. The application of the cocaine may not relieve it entirely, and the application of the hot boracic acid may likewise fail, but they will do some good. An anodyne will bring the case to the subsequent use of the saline purge, which will achieve the final result.

I wish to say explicitly that, after the employment of a great many cathartics, I have come to the conclusion that Epsom salts is by far the best.

When, in the case of children, the drum has become perforated and the disease runs into a purulent inflammation of the middle ear, measures should be employed which are not within the limits of this article.

To sum up my views upon this subject, I may say that the treat-

ment of acute otitis media is local and general; that the local treatment is the employment of hot cocaine solution, the instillation at frequent intervals of hot boracic acid, and the application, possibly, of a hot-water bag to the side of the head; and that the use of paracentesis is a possible method when there is great bulging of the drum. The general treatment consists in the exhibition of Epsom salts in drastic doses, and of an anodyne in the case of children.

## THE OPERATIVE TREATMENT OF SCLEROTIC CATARRH OF THE MIDDLE EAR.

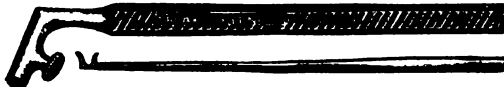
CLINICAL LECTURE DELIVERED AT THE CHICAGO POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL.

BY SETH SCOTT BISHOP, B.S., M.D., LL.D.,

Professor of Diseases of the Nose, Throat, and Ear in the Illinois Medical College;  
Consulting Surgeon to the Mary Thompson Hospital, etc., Chicago, Illinois.

GENTLEMEN,—I show you an instrument called an ossicle vibrator, which I have devised for the purpose of breaking up adhesions in the middle ear, and ankylosis of the ossicles. (Fig. 1.) It consists of a shaft of steel armed with two little levers at the distal end, and fashioned at the proximal extremity to fit into the angular handle of the middle-ear instruments. It is used in this manner:

FIG. 1.



Bishop's ossicle vibrator.

An incision is made through the drum-head close to the anterior border of the hammer-handle and parallel with it from the short process to its tip, under cocaine anæsthesia. Then the distal lever, which is curved for the purpose, is carried through this slit and behind the mallet, when the handle falls between the two little levers. Then they are slipped along upward, embracing the handle, until the stronger part of the bone is reached and the levers fit the handle somewhat closely. Now the retracted mallet is slowly and very gently drawn upon until it is felt to move, or until the adhesions are felt to give way, and to the extent of bringing the handle to its normal position. The greatest care must be taken or the adhesions may give way very suddenly with a jerk and the mallet might possibly be dislocated, or the handle might be fractured, especially if

the instrument were allowed to slide downward upon the weaker portion of the handle. I have not known these accidents to attend the use of my instrument, but one can conceive that they are within the range of possibilities. Again, a patient has become pale just as the adhesions yielded to the traction, and nearly fainted. This was probably due to the disturbance of the intra-labyrinthal fluid as exaggerated motion was effected in the stirrup. Some most remarkably beneficial results have followed the use of this simple method of mobilization of the ossicles. No harm has been known to arise from it. After making the incision and before introducing the vibrator, it conduces to the comfort of the patient to instil a few drops of a warm ten-per-cent. cocaine solution.

Incision of the posterior fold of the drum-head is indicated when there is great sinking inward of the membrane, with foreshortening of the handle of the mallet, prominence of the short process, and a stretched appearance of the membrane about it. This condition, associated with serious impairment of hearing and head-noises that are unimproved by the treatment already detailed, calls for this simple operation. The section is best made about midway in the fold, and the knife (Fig. 3, No. 2) is made to cut from above downward, with care that it is not carried deeper than is required to sever the fold, otherwise the chorda tympani may be severed, producing paralysis of taste.

Although I have made such sections frequently, I have never known this to follow, but such results have been reported. Patients generally observe a sense of relief from pressure, clearness in the head, diminishing of subjective noises, and sometimes improvement in hearing. In the class of cases in which I have mostly practised this operation I have not been able to follow up the results for a number of years, but have known the benefit in a few to persist for several years. In others of a worse type the improvement has been transient.

Multiple incisions of the drum-head have proven beneficial in some instances. In 1886 I reported the results of a series of cases to the meeting of the American Medical Association, from which I will quote: "For the purpose of making a crucial test of the efficacy of this procedure, I have made it the last resort in those cases that afforded no hope for relief from any other treatment. Perhaps the propriety of operating on those patients that seemed to promise

no results might be questioned, were it not for the facts that in nearly all of them there was an unexpected improvement, and that no unfortunate consequences followed the operation. The cases I have chosen to operate on were far more hopeless than those with chronic suppurative inflammation. The consideration that the former respond so little to our efforts, while the latter are so amenable to treatment with inflations, cleansing, dioxide of hydrogen, boracic acid, bichloride of mercury, etc., with the result of not only arresting the disease but of improving the hearing, has led me to seriously contemplate the advisability of establishing the suppurative process in sclerotic inflammation of the middle ear. In three cases, only, in my practice has this condition followed the procedure under discussion, and the results in the series of cases reported were satisfactory, especially when it is considered that they were the most unpromising and had proven the most intractable to the usual methods of treatment. But as remarked before, this experimental work, which was carried out mostly in dispensary practice, did not afford opportunities to follow up the results for a number of consecutive years. The simple incision, of course, closed in a few days, but the tension of the drum-head apparently was restored to more nearly the normal." At the present time (April 12, 1898) one of these cases has again fallen under my observation, and shows that the really brilliant results obtained twelve years ago have persisted during the intervening years. The patient, who is an intelligent real estate dealer, gives an excellent account of his case.

Another method that I have since pursued with considerable success is the excision of areas of the drum-head, usually triangular in shape. Under cocaine I make triangular flaps with the apex above, and then sever the attached base, removing this piece of the membrane entirely. It was sometimes easiest, after making the two sides of the triangle, to grasp the apex with delicate forceps in one hand while the base incision was made with the other. The improvement in some patients in whom there was no labyrinthal disease was very gratifying, and in private patients I was able to demonstrate the possibility of maintaining the aperture for a considerable time. In one instance it had remained open a year and a half, when the patient removed from the State. I had a peculiar experience with the opposite ear. The first operation afforded so much improvement that he requested that the same operation be

performed on his right ear. It was done, and a slight, mucopurulent discharge followed, but soon ceased. While the discharge lasted, the hearing was considerably improved and the tinnitus relieved. After the discharge ceased, the hearing began to diminish, when he expressed regret that the ear had not continued moist. This led me to moisten it with warm, pure vaseline, but when I removed it a few days later the very large perforation was entirely closed.

The removal of sections of the drum-membrane may prove otherwise advantageous. It affords accessibility to the tympanic cavity for the instillation of various remedies and the destruction of adhesions, and it reveals whether the entire resection of the drum-head would improve the hearing. In case the membrane is so thickened and sclerosed and infiltrated with calcareous deposits as to preclude the possibility of its responding to any except extraordinary sound-waves, and the labyrinth is not involved, the opening of a window in the drum-head will admit sound to the stirrup and to the round window and prove whether the entire absence of the membrane would prove remedial. If the adhesive process has not ankylosed the stirrup in the oval window, or invaded the round window, vibrations can then reach the labyrinth if the barrier to their admission be removed. I have employed this test to determine whether excision of the entire drum-head would afford successful results.

Division of the tensor tympani tendon is not much in favor among American aurists. The indications for it are not clearly defined, and the appearances that suggest the shortening of this muscle, retraction of the membrana tympani and foreshortening of the mallet-handle, are also just as characteristic of the presence of membranous folds and bands of adhesion. The results of tenotomy have been either so unsatisfactory or so positively deleterious that the operation is not encouraging.

#### EXCISION OF THE MEMBRANA TYMPANI AND OSSICLES.

This operation for sclerosis is a subject concerning which there is probably less unanimity of opinion among otologists than upon any other. While a few American aurists, especially Burnett, Sexton, Blake, and Jack, have been enthusiastic advocates of the operation, and some others have followed their lead for a time, the majority appear to have receded to a more conservative position. At

the meeting of the Section of Otology at the Tenth International Medical Congress in Berlin in 1890, the Continental leaders in this specialty expressed themselves in extremely conservative terms on these subjects. Several years ago, through the columns of the *Journal of the American Medical Association*, I invited all who had performed this operation to communicate the results for the purpose of publishing a collection of experiences that would afford a just estimate of the average value of this operation. The responses were so few and so unsatisfactory as to force the conclusion that the operation was either little practised or was disappointing. There is probably little or no diversity of opinion concerning the utility of the operation in suppuration of the middle ear, especially when there is ossicular necrosis; but as practised for sclerosis there has been so much diversity of opinion and sad, disappointing experiences reported during the past ten years, that candor requires that the subject be treated with reference to the bad as well as the good results. A number of cases have been under my observation, upon whom the operation has been performed by surgeons both East and West, with the effects of producing a suppuration of the middle ear, destroying the hearing, apparently intensifying the noises, and producing more or less vertigo. At the present time I have under treatment a physician from a far-Western State, whose ossicles were removed from one ear by a prominent aural surgeon several years ago. All the ill results enumerated followed the operation, and although the hearing was two inches for the watch before the operation, that ear has been totally deaf ever since, and the opposite ear has deteriorated. This is a fair type of numerous similar instances that have come to my personal knowledge and under the observation of other physicians who have been kind enough to report them to me. Würdemann had the courage to report several similar results at the meeting of the American Medical Association in 1892. It is worthy of mention that nearly all of these unfortunate cases were operated upon by specialists in eye and ear diseases, so that the results cannot be attributed to the want of familiarity with the subject. It is not my purpose to inveigh against this procedure as an operation, but to emphasize the necessity not only of the utmost precision and gentleness in operating, but also the most painstaking preliminary examination and experiments to determine the possibility or otherwise of beneficial results. For example, if the hearing-tests demon-

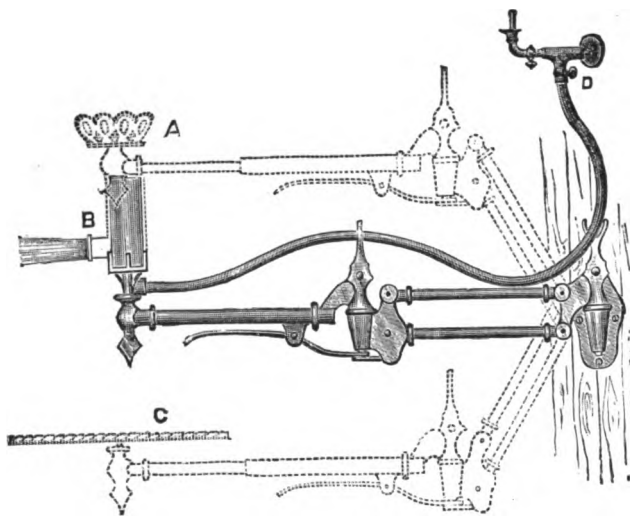


strate that the labyrinth is involved in the disease, the inutility of the operation is established. If no improvement follows a resection of a portion of the drum-head so as to admit sound-waves to the fenestræ leading to the internal ear, no help can be expected from excision of the whole membrane. We do not lose sight of the fact that, by removing the drum-membrane and the two larger ossicles, we are afforded access to the stirrup so as to mobilize it. Some advantage certainly is to be conceded to this measure, although mobilization of the stirrup is not as simple an act as one might believe. Even with every vestige of the membrane removed, the stirrup is situated so high that a good view of it is difficult to obtain, and I have found it easy to dislocate when it was not ankylosed.

#### OPERATION FOR EXCISION OF THE OSSICLES.

The ear should be prepared by syringing with a warm solution of bichloride of mercury, one to one thousand, and the instruments

FIG. 2.

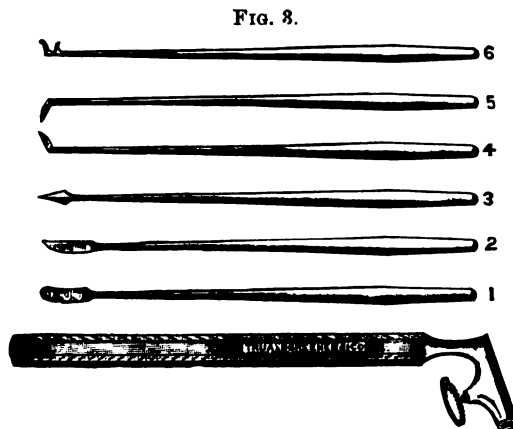


Bishop's adjustable lamp-bracket.

should be immersed for three minutes in boiling soda water. For several years past I have used ether to the exclusion of chloroform, instructing the anæsthetizer to administer only so much as is absolutely necessary to secure quiet and freedom from suffering. Cocaine anæsthesia is not as effective as ether. After removing all

*débris* of any nature from the canal, it is dried and closed with absorbent cotton until the operation commences. If ether is used, the patient must occupy a recumbent position. I use tables high enough to bring the patient's ear opposite my eye as I sit facing the table. A brilliant illumination is needed. I have used mostly the Argand gas-lamp and light-condenser. (Fig. 2.) One will have a clearer view of the field of operation if the room is darkened so that no light penetrates the operator's eye except that reflected from the ear cavity.

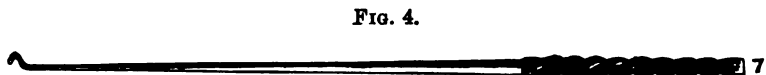
The instruments necessary (Fig. 3) are a paracentesis knife, No. 2, a blunt-pointed bistoury, No. 1, two angular knives, right



Bishop's middle-ear instruments.

and left, Nos. 4 and 5, two ossicle hooks, right and left (Fig. 4), a pincette (Fig. 5), a dozen cotton carriers armed with cotton, a quart of hot, sterilized water, and a syringe.

The operation proceeds as follows: The drum-head is incised with knife No. 2 near the periphery behind the short process of the



Bishop's ossicle hook.

mallet. Into this opening the blunt-pointed knife, No. 1, is inserted and carried first below, then sweeping the lower, and the anterior attachments until the roof is reached; then this attachment is

severed until the whole circular incision is completed, ending at the first entrance. The knife is best carried first from above downward, for the reason that less hemorrhage is likely to obstruct the view than if the more vascular *membrana flaccida* were first cut. There is less hemorrhage, also, if the knife is kept a little way from the periphery of the membrane. Now the angular knife is used to separate the articulation of the anvil and stirrup. The anvil is extracted by aid of the hooked probe, and the attachments of the mallet are then divided, when it is brought away with the pincette.

The operation is a very short one, requiring but a few minutes ordinarily if there is not much hemorrhage, or if the adhesions are not embarrassing. Rapid use of the cotton carriers, which should

FIG. 5.



Poltzer's pincette.

be kept prepared by a nurse, will keep the field quite clear ; but in cases of considerable bleeding the syringe and quite hot water can be brought into requisition. It is difficult to avoid severing the *chorda tympani*, but the resulting paralysis of taste is of short duration. The ear cavity should be dried after bleeding has ceased, covered with a layer of aristol from the small powder-blower, and the canal closed with iodoform gauze. While there is considerable reaction in some cases, followed by discharges of a mucopurulent character, in others there is little or no disturbance. The patient should be kept quiet and his diet restricted until healing takes place. By properly restricting the diet both before and after the operation there is less tendency to regeneration of the drum-head. The latter occurrence is quite frequent. In the case of the physician men-

tioned, there is a false drum-head which I have not removed, for the reason that no possible good could come of it.

In another case of a very robust man from Kansas I removed the third adventitious membrana tympani, at his request. In the spring of 1893 a surgeon had removed his drum-head and mallet. In seven days after the operation he says the drum-head had been reproduced. This was removed, and in seven days more the surgeon said that another had closed the tympanum. A third operation was had, and in fourteen days another drum-head had formed. Two years afterwards the patient came to me with the request that I remove this remaining fourth drum-head with which nature had supplied him. He suffered from great tinnitus and uncomfortable sensations of pressure, etc. Examination revealed labyrinthal involvement, and I discouraged the procedure. But, notwithstanding the assurance that no improvement was to be expected, the patient insisted upon the operation with the hope that it might afford some relief to the tinnitus and pressure symptoms. Therefore I removed the drum-membrane and anvil before the class in this school, June 21, 1895, and cauterized the periphery of the drum-head so as to completely destroy the whole circular attachment. A few days afterwards I found the stirrup dislocated, and removed it. No unfavorable symptoms followed; the membrane has not been reproduced, and the slight discharge following the operation soon ceased. The ear has remained in good condition ever since, but, although the patient imagined himself better, I could discern no improvement. The tinnitus and other symptoms were not removed or considerably improved. The patient thought he could hear, but accurate tests proved the contrary. The case is instructive in showing that thorough galvano-cauterizing the peripheral attachment of the membrane will prevent its regeneration. I do not often employ this cautery in the ear on account of the great heat generated in such a minute, enclosed space, but the chromic acid has too superficial an effect to accomplish the purpose.

While I mention these unfavorable cases, and I might cite others who have come under my care, one of whom is the most distinguished of American editors, it is not for the purpose of condemning the operation itself, for I believe that these unfortunate results are attributable either to an unwise selection of cases or to unforeseen accidents attending the operation. For example, why should two inches of hear-

ing for the watch be exchanged for total deafness, vertigo, etc.? The results point towards an injury to at least one of the fenestræ opening into the labyrinth. But the reverse of this picture presents some excellent and even brilliant results. Some cases that have proved intractable to the usual measures have yielded to this; but these are the ones in which the labyrinth has not been involved, and the adhesive process has not destroyed the usefulness of the stirrup and membrane of the round window, and in which excision of a small section of the membrana tympani would demonstrate the possibilities of the operation.

Mobilization of the stirrup has been practised with favorable consequences, especially by Jack; but the crura of the stirrup are so exceedingly delicate and fragile that they are quite likely to break on applying side pressure to them or on traction with the hook. This manœuvre is not in favor with otologists generally. After the membrana tympani has been removed for sclerosis the conditions are most favorable for mobilization. The probe can then be introduced alongside the stirrup and pressure exerted in all directions to break up adhesions and effect mobility. The hook can then be engaged in the apex of the converging legs of the bonelet and drawn upon until slight motion is had. But if the adhesion gives way suddenly the stirrup will be dislodged or extracted unless great care is exercised.

# Medicine.

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## SOME FORMS OF GASTRALGIA.<sup>1</sup>

CLINICAL LECTURE DELIVERED IN THE AUGUSTA HOSPITAL.

BY PROFESSOR C. A. EWALD,

Director of the Augusta Hospital, Berlin, and Professor of Diseases of the Digestive Tract at the University of Berlin, Germany.

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GENTLEMEN,—I showed you yesterday five patients, four men and one woman, whose principal symptom was cramp-like pains in the region of the stomach, a symptom which some years ago, before modern methods of investigation in stomach diseases had enabled us to differentiate the various conditions which gave rise to it, was treated of in the text-books as an affection by itself, under the name of gastralgia or cardialgia, because of the location of the pain in the cardia, or finally as gastric cramp, from the character of the pain. We have learned that we have to do in such cases with very varying pathological conditions, and it is some of these that I propose to discuss before you this morning, with our five patients of yesterday and samples of their stomach contents as a text.

Our first patient was a man who is being treated in the polyclinic (out-patient department), and who came complaining of “cutting” pains in his stomach region and attacks of vomiting, for which he knew no cause. They were not preceded by the eating of anything that disagreed with him, and sometimes came on, as in the morning, when his stomach should be empty. He brought some of the vomited matter with him, and it had a brownish-green tinge to it. Tests for the presence of blood in the vomit, including the spectroscopic examination, showed that blood-coloring matter was present.

We then passed a stomach-tube in your presence, and in wash-

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<sup>1</sup> Reported by Dr. James J. Walsh, Berlin, Germany.

ing out the stomach got what seemed to be such an amount of blood that some of you at least, I think, must have been a little alarmed. I reassured you, however, that it was only a usual symptom of the affection that was present, and that that was not ulcer of the stomach, so that there was no cause for alarm. After the lavage, careful palpation of the stomach region gave us no points of diagnostic value. The fifty cubic centimetres of greenish stomach contents showed us, after careful examination, that the gastric chemism was normal.

Our second patient is an old friend of ours; he has been in the hospital eleven times during the last five years, and his case is well known. He comes for cramp-like pains in the stomach region, often localized in the *scrobiculus cordis*, though sometimes beginning lower down in his abdomen in the region of the bladder and mounting upward. His attacks are accompanied by vomiting, too, and he vomits the greenish-blue liquid you see here. The vomited material is often very acid, but sometimes not more than normally so, and exceptionally has been found to be neutral.

Our first patient gave us a history of having had recurring attacks of pain in his stomach for some years. At first they occurred not oftener than three or four times a year, but of late they have been coming more frequently. Our second patient gives us a similar history. At first the pains occurred every two or three months, then once a month, then nearly every week, and now he is seldom free for even so long an interval as this. This history is characteristic of the disease, and so I call your special attention to it.

The third patient is a woman who often complains of pains in her stomach, the severity of which you can judge for yourself, as it was she you heard complaining so loudly and so bitterly as you passed through the hall this morning. Details of her history are not easy to obtain from her owing to her psychic condition, so we shall see what her stomach contents tell us. They are not green or greenish blue, as in the other cases, but yellow and cloudy. This appearance is not due to any remains of undigested food, or recently ingested food particles, since we obtained the gastric contents from her stomach after she had been fasting overnight.

The test with congo paper shows that no hydrochloric acid is present, though litmus paper shows that there is a slight acid reaction. I shall filter the stomach contents and test it for lactic acid, though I do not expect to find any, as our tests beforehand always

failed to show it. The slight acidity noted is due to the presence of minute quantities of various organic acids, no one of which is present in sufficient quantity to give a characteristic reaction.

Our fourth patient came to us three days ago because of crampy pains in the stomach region, which were followed by attacks of vomiting. Yesterday he vomited in a single spell the large quantity of material that you see in this glass jar. There is more than two and one-half litres, or five pints, of mingled fluid and solid contents, though the solid material is in the shape of rather fine particles. On standing it has as you see separated into three layers,—an upper layer of foamy consistence, a middle fluid layer, and at the bottom of the glass a sediment layer of slime-like material. The color is different for the various layers,—whitish yellow at the top, a dirty cloudy yellow in the middle, a lighter yellow at the bottom.

The test with congo paper shows the presence of plentiful hydrochloric acid. Tests for lactic acid show that it is absent, as might be expected with the abundant hydrochloric acid present, for lactic acid is always the indication of abnormal fermentative processes in the stomach, and these do not take place in the presence of free hydrochloric acid. Microscopic examination of the sediment, as you can easily convince yourselves by looking at the slide at present under this microscope, shows the presence of sarcinæ, of yeast-cells, of amyloid bodies, and of small spherical fat particles.

It required, as you may remember yesterday, very little effort and the application of very little siphonage to get a considerably larger quantity of liquid contents out of his stomach, after he had been fasting overnight, than would be found normally. Usually ten to twenty, at most fifty, centimetres, or three to six drachms and at most two ounces, of fluid can be obtained from a healthy stomach in the morning. Here we got without any difficulty four hundred to five hundred cubic centimetres, or fifteen to nineteen ounces, of this greenish fluid, which has in the mean time, as you see, let fall a sediment. This sediment we will proceed to have the assistant put under a microscope, and we shall see it later. In this fluid also we find free hydrochloric acid.

With as large an amount of vomit as is here the case, and with the presence of sarcinæ and yeast-cells, as well as the amyloid and fat bodies, we must think of stagnation of the stomach contents and dilatation of the stomach. We tried to demonstrate that yesterday



by pumping his stomach full of air, so as to bring out clearly the outlines of the stomach on the abdominal walls. But his stomach is evidently in an irritable condition, and he himself is nervous and restless. Despite every effort of dissuasion, he would so contract his abdominal muscles that as soon as sufficient air had been forced into his stomach to distend it somewhat it would be forced out again by the tense contraction of his belly walls. We shall to-day pass an electric light into his stomach, and by gastrodiaaphany find the outlines of it.

After filling the stomach well with water we pass the electric light and this rubber tube, in which are contained the conducting wires, just as we do the stomach-tube. Then we turn on the current. At first we get the red shimmer only at the cardiac region, so that the lamp needs to be pushed farther into the stomach. We turn on the current again, and see nothing,—just the faintest shimmer, very distant. In dilated stomachs especially, the lamp would sometimes seem to get directly behind the tube, the shadow of which thrown on the anterior wall of the stomach conceals the outlines of it. Here at the sides comes a shimmer of red light.

We manipulate the conductor a little to get the kink out of the tube, and now we see the outlines of the stomach very clearly. It is, as you see, enlarged, though not very much so, and extends downward only to the navel. There is a suspicious irregularity about the outline of the greater curvature that makes me inquire of him when he last had a passage from his bowels. He says not since his entrance three days ago. As a filled, distended transverse colon might by its presence keep the dilated stomach from assuming the position of dependence that it otherwise would, we cannot draw any sure conclusions as to the dilatation of the stomach in this case.

The patient was given the ordinary hospital cathartic yesterday, but it failed to operate, and an efficient purgative will have to be given him now, and at our next clinic we will be able to decide just how much dilatation of the stomach exists. In the mean time I do not think that there is very pronounced dilatation in the case. His attacks of pain and vomiting come at rather long intervals, so that I do not think there is any question of any permanent stenotic constriction of the pylorus, or of a neoplasm hindering the outflow from the stomach and so leading to the collection of large quantities of material in the organ. I incline to the view that we have to do with something periodic in character in the nature of a cramp-like con-

traction. Palpation and inspection, I need not say, have been of no help in the case.

Our fifth patient is a man who comes complaining, like the others, of crampy pains in the stomach. His attacks are, however, not so severe as in the other cases. They do not have distinct intervals of intermission, but occur almost daily. They are not immediately dependent on the taking of food, nor do they occur at any definite time after the food has been taken, yet they bear some relation to the presence of irritating substances in his stomach, as will be clearer after you hear more of the case. Of late his pains have let up considerably, owing to a special *régime* to which he is subjected, and of which I shall speak a little later; only at night now is he bothered with pain.

The fluid we obtain by the stomach-tube from his stomach in a fasting (overnight) state is not large in amount, is of clear watery color, containing flocculi of mucin evidently from sputum or nasal secretions swallowed during the night. It contains one pro mille of hydrochloric acid, and has a total acidity of 80; that is, the acidity of one hundred cubic centimetres, or three and a half ounces, of stomach contents completely neutralizes eighty cubic centimetres, or two and three-quarter ounces, of a decinormal alkali solution.

Here, then, are our five cases, in which pain in the stomach region is the main symptom and *the* complaint with which they come to the doctor for treatment. Are they all due to the same cause, or are they entirely different affections? Any rational treatment and our prognosis of the different cases must depend on how far we can answer this question. The differentiation of the cases is of the utmost importance.

Almost without any difficulty the further examination of our patients causes them to fall into two groups. The symptoms that guide us in this division of the cases, however, have nothing to do with the stomach, and emphasize the necessity that always exists in stomach cases,—of thoroughly going over the patient, of diagnosing, in a word, the patient's general condition as well as the symptoms of the special ailment complained of.

The first two male patients and the woman fall into one group, because of certain nervous symptoms in the cases. All of them have unequal pupils, with the Argyll-Robertson phenomenon,—reaction to accommodation but not to light. In the legs of the first patient there are some easily demonstrable areas of disturbed sensi-

bility, mainly showing marked anæsthesia. The woman has certain psychic symptoms, which have led her friends to apply for her admission into an asylum for the insane. She has besides a tremor of the lips when she speaks, her speech is somewhat scanning in character, with an almost typical tendency to the elision of syllables at times. It is an almost classical picture of the first stage of progressive paralysis of the insane.

The male patients, though neither of them have symptoms of incoördination as yet, are in the incipient stage of *tabes dorsalis*. Duchenne first pointed out the occurrence of stomach cramps, crises gastriques in *tabes*, and the subject was further studied by Delamarre, the French clinician. Our exact clinical knowledge of them, however, we owe to Charcot and his school. Charcot and his pupils pointed out that the lightning pains of *tabes*, of which the stomach cramps were but one manifestation, might occur in connection with almost any of the nerve tracts in the body. The region supplied by the vagus was, under certain not very well understood circumstances, more liable to suffer than those supplied by others of the cranial nerves. Laryngeal, œsophageal, and cardiac crises were to be noted as well as the gastric and anal crises. Renal crises, apart from any suspicion of renal calculus, occurred.

A certain favoritism for the digestive tract was remarked, especially the two abdominal ends of it, the stomach and the anus. The stomach crises have a very characteristic symptom complex. They either come on suddenly, justifying the name lightning pains, with intense excruciating pain in the bladder region or in the inguinal regions, gradually mounting to the epigastric region, where they reach their acme, or they begin with a feeling of uncomfortable-ness in the epigastrium, which becomes painful and then tearing in character. The pain is followed by vomiting, usually, as here, of green or greenish-blue fluid, often mingled with a little blood, sometimes accompanied by large and almost startling hæmatemesis.

They may last twenty-four or even forty-eight or seventy-two hours, though they may be over in a much shorter time. Characteristic of them is that immediately after they cease the patient feels perfectly well. If the gastric crisis has disturbed the regular taking of food, the patient usually at once manifests a good appetite and proceeds to eat a hearty meal.

The crises come on at intervals, just as our two male patients

have described, which grow gradually shorter, at first every four or five months perhaps, then six times a year, then every month or every week, and even oftener. Their frequency seems to bear no relation to any cause we have been able to discover. Patients sometimes attribute them to indiscretions of diet, but this would seem to be the result of the desire to find some explanation for them, as they occur also in hospital patients under a well-regulated dietary.

Their increase in frequency would seem to correspond to the advance of the disease of the central nervous system to which they are due. As the gray degeneration of the posterior columns of the cord becomes more pronounced, or rather as Landouzy, Déjerine, and Kahler and Oppenheim have shown, as the degeneration of the nucleus of the vagus advances, the intervals become shorter between the crises.

Usually, however, the gastric crises occur in the preataxic stage of tabes,—i.e., before symptoms of incoördination have developed. I have often had patients in private practice who have not had a single symptom of tabes except the characteristic gastric crises.

Our second patient of to-day we had known here at the hospital for four years before any additional symptom of tabes developed. During this last year he has developed inequality of the pupils and the Argyll-Robertson symptom, enough, with his gastric crises, to stamp his case as surely tabes dorsalis, though he has not the slightest incoördination, has his knee-jerks preserved, can stand with his eyes closed and his heels and toes together without wavering, and has no paræsthesiæ and no anæsthesia or other disturbances of sensibility.

In such incipient cases, where there are only the gastric crises and no other typical confirmatory symptoms, the differential diagnosis is, as may well be imagined, often extremely difficult. One must think, in such cases of lead-poisoning, of morphinism and of other neuroses of the stomach, neurasthenia or hysteria, which at times cause a set of symptoms not unlike the gastric crises of tabes.

For the differential diagnosis of lead-poisoning there would be the history of working in the metal or in some industry in which it was used. There would be, perhaps, the blue line on the gums and the anæmia of saturnism, or some neurotic symptoms, especially in the region supplied by the radialis. There would be a history of long-standing obstinate constipation, and the pains would be localized lower down in the abdomen around or below the navel.

For morphinism, for this too often gives a series of gastric crises not unlike tabetic pains, there would be the history which I need not tell you must not be gotten from the patient himself, as morphine *habitués* are absolutely unreliable in personal statements as to their habits with regard to the drug. Then there would be the symptoms of the intoxication, the tremor, the failure of memory, the alternating stages of depression and exaltation when under the influence of the drug and without it, the contracted pupils, which must not be mistaken for the pin-point pupils of tabes, and, finally, the generally run down condition. In neither of these intoxications, lead or morphine, do the attacks come suddenly in the midst of perfect health and disappear, to have the patient return at once to his wonted appetite, as is so characteristic of tabes.

As to neurasthenia and hysteria, we will speak of them later in discussing the other two cases. In deciding for the presence of tabes in these cases, a history of syphilis in the patient some years before will be confirmatory of suspicions raised by pains in the epigastrium which simulate gastric crises. Unfortunately, here, unlike most syphilitic affections, the therapeutic test will be of no help, for while tabes would seem in most cases to develop in patients who have had syphilis, antisyphilitic treatment will not modify the course of the symptoms.

I have said that the pains grew worse as the tabes progressed, but this is only true up to a certain point. The nervous degeneration in the sensory columns finally seems to reach a stage where painful sensations are no longer easily aroused, and then the gastric crises become less frequent. I have a patient now who has been under observation for ten years, who began with the gastric crises and absolutely no other tabetic symptoms. Gradually the attacks became more frequent, and then frank tabetic symptoms developed. He has now become almost completely paraplegic from intense incoördination, but he has had no gastric crisis for years.

It is to be remembered that at times the anal crises alternate with the gastric attacks. There comes tenesmus, but efforts to evacuate the bowels are futile. This may last for some minutes or an hour, sometimes longer. When an evacuation is finally effected, it is usually one or more scybalous masses, dry and hard, though such a stool is often followed by relief. Another perhaps more frequent form of anal crisis is accompanied by intensely painful sensations in the region of the anus, and the patient complains of a knife or

stick being forcibly pushed into his anus and turned now this way, now that. Like the stomach crises, these are sometimes the first symptom of tabes; like them, too, they are apt to be more frequent as the disease advances, until serious ataxic symptoms have developed, when they usually remit.

We have spoken of these gastric crises always as if they were exclusively tabetic in origin. The woman patient, however, is suffering from general paralysis. In a wider sense this is considered by many to be a tabetic process, involving the brain, so that the connection is easy of explanation. Gastric crises may occur, however, also in multiple sclerosis, and in general, if we are to trust the ordinarily accepted pathology of them, in any affection leading to degeneration of the vagus nuclei.

The chemism of the stomach in these cases is very interesting. Sahli advanced the opinion that the crises were due to an increase of the acidity of the stomach, but this has not been confirmed by further observation. In our hospital experience here we have found the most varying conditions of stomach acidity before, during, and after the crises,—hyperchlorhydria, normal acidity, and achlorhydria. We have a striking example of this in the three patients under consideration. In the woman free hydrochloric acid is absent; in one of the men it is about normal; in the other abnormally increased. In two of the cases the stomach contents are of a greenish-blue tint. This used to be thought due to bile; but the coloring matter is not bile, though we do not know quite what its origin is.

The therapeutics of gastric crises are most unsatisfactory. The ordinary narcotics—pilsenkraut, codeine, laudanum, or even morphine—given by the mouth fail to give relief, as a rule. Morphine subcutaneously is usually effective, but as the disease is essentially chronic, there is the greatest danger of forming the opium habit. Never, never allow the patient to have a syringe and give himself injections. I cannot insist too much on that.

It is to be remembered, too, as you saw from our discussion of the differential diagnosis of gastric crises, that there is a form of stomach cramps which supervenes on the use of morphine, and which makes a most pitiable complication. No more pitiable object can well be imagined, however, than a tabetic become morphinist, and the intoxication undoubtedly hastens the degenerative process in the cord and shortens life by its effect on nutrition. We cannot absolutely refuse morphine, but its administration must be put off

as long as possible, then carefully regulated, and the patient must be kept thoroughly under the physician's control.

From lavage, electricity, or hot or cold applications, during a severe gastric crisis, I have never seen any good results, though they are of some help perhaps in mild attacks. As to the antipyretic analgesics, antipyrin, phenacetin, etc., the same thing holds,—they fail of effect in severe cases, though they are useful additions to the therapeutics of the milder cases.

With the treatment for the *tabes dorsalis* itself we have had no success. We tried the suspension treatment in a large number of cases here at the hospital. For the first few treatments patients claimed to have obtained distinct relief from this method, but as soon as the effect of the novelty of the treatment had worn off, and suggestion failed to play her usual *rôle* in the matter, then all the old symptoms returned with their wonted intensity.

From the use of mercury and the iodides I have never had any good results. The etiological connection between *tabes* and syphilis in most cases naturally makes one turn to antisiphilitic treatment. We have tried very thoroughly the *schmier kur* (rubbing of mercury, usually in the shape of gray salve, into the skin) here at the Augusta Hospital. In severe cases I have had six grains (about one and one-half drachms) rubbed in each day, until over one hundred grains had been used, but to no purpose. With time and the progress of the disease the crises will become less and less frequent. In the mean time the careful use of morphine subcutaneously seems to be the only thing that helps.

Of our five patients, there are two of whom we have not yet spoken. They present no nervous symptoms, and cannot be classed with the other cases. I may insert here that occasionally cases of periodic stomach cramps occur for which we can find no cause; sometimes these recur at regular times each year, which would make one suspect that they had some connection either with changes of temperature or with variations in food which are sometimes quite marked for certain people at different seasons of the year.

In neither of these cases shall we have recourse to any such indefinite explanation. In the patient from whom this large amount of vomit comes, and from whose stomach while still fasting we got these five hundred cubic centimetres, or nineteen ounces, of slightly yellowish-green fluid, there is evidently the condition that I have described as *parasecretion*. Others have called it *hypersecretion*, or

hyperchlorhydria. Owing to irritation there is a secretion into the stomach of gastric juice, even at a time when there is no food in it to be digested.

This is always due to nervous influence, though the irritation of the nerves may take place either through the peripheral nerve-endings themselves in the stomach walls, or come from the central nervous system. The presence of a gastric ulcer or of its products in the stomach may by irritation provoke this neurotic para- or hypersecretion, or other changes in the mucous membrane, as the desquamation of its upper cellular layers or slight erosions of the mucous membrane, may expose nerve-endings to such irritation. On the other hand, it may occur as a pure neurosis, and such it seems to be here. We have no reason to diagnose ulcer of the stomach from the history of the case, or the presence of blood in the vomit, or a localized area of tenderness in the stomach region, or the occurrence of pain shortly after eating, or especially after eating acid or spiced foods. We have none of these symptoms. The pains seem to be due to the collection of food and liquid in the stomach, and this is due to an irritative contraction of the pylorus.

The therapy of the case will be the gradual building up of the lowered tone of the nervous system and the avoidance of too much food at any one time, but small amounts more frequently. Gastrodiaphany showed us in this case, I think, that there is no serious dilatation of the stomach, though our study of the case is not complete.

[NOTE.—This patient was put under the same *régime* as will be seen later to have been successful with the next (the fifth patient). All nourishment by the mouth was withdrawn for six days, and after two weeks his condition had so much improved that he was able to leave the hospital practically cured. Under the judicious use of nutritive enemata in such cases very little suffering is inflicted on the patient, less than the continuance of their pains, because of the irritation due to the presence of food in the stomach, and the therapeutic effect of six or seven days of absolute rest for the stomach—the capital indication of the case—is extremely satisfactory.]

Our fifth patient, from whom we obtained this watery-colored fluid with the mucin flocculi in it, and which gives the proper reactions and digests albumen, is suffering from chronic gastritis. There is a nervous element in the case, too, but it is very different from the preceding one. He has been a drinker, and the conse-



quence has been hyperæmia and desquamation of the mucous cells of his stomach, so that the nerve-endings have been rendered extremely irritable.

There are erosions of the gastric mucous membrane in consequence of the chronic catarrh, and sometimes there are streaks of fresh blood in the contents of his stomach, or bits of mucous membrane with the cells in the well-known stage of cloudy swelling. The patient has lost forty-five pounds, one-fourth his weight, in four months, owing to his inability to take food.

Here the indication is rest, and having found that even the best regulated liquid diet still caused irritation and pain, we have for the past week fed him entirely by the rectum. As a result of this his pain has almost completely left him. He still complains some at night, the reason being obviously that the swallowing of oral and nasal secretions leads to parasecretion, and so to irritation of gastric peripheral nerve-endings. After lavage in the morning there is no more discomfort. Interestingly enough, he has no sensation of hunger, despite the fact that for a week he has taken no nourishment by the mouth. This is sometimes the case in rectal feeding, but oftener the hunger has to be stilled by cocaine, which is the best drug for the purpose. As to just how much albumen is absorbed from our nutritive enemata I cannot say, despite the amount of work that has been done in the matter. Patients always decrease in weight, but it is certain that the clysters are of real benefit, and that an absorption that corresponds nearly to the metabolic products of the organism can be effected through the intestine. In these cases of irritable chronic gastritis nutritive enemata form our best method of treatment, since they enable us to fulfil the most striking indication of the case,—the stomach's crying need for absolute rest. Yesterday, after seven days of rectal feeding, we gave him some milk, which was well borne and led to no complaints of pain. Afterwards we gave him some mashed potatoes, but his stomach is not yet ready even for anything as bland as this, so there was discomfort. We shall keep him on an absolute milk diet for some time, and report the results.

Now, gentlemen, we have discussed the five interesting cases that our large service here at the hospital has enabled us to put side by side. I hope that they have given you a clearer insight into that knotty chapter, the gastric neuroses, and especially that multi-significant symptom, recurring attacks of cramp-like pain.

## BRONCHOPNEUMONIA FOLLOWING MEASLES.

CLINICAL LECTURE DELIVERED AT THE HOSPITAL COLLEGE OF MEDICINE.

BY JOHN A. LARRABEE, M.D.,

President of the Faculty and Clinical Lecturer on Diseases of Children in the Hospital College of Medicine, Louisville, Kentucky.

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GENTLEMEN,—This little patient, a male, aged ten months, is brought to us for examination and treatment by the mother to-day for the first time. We are told by the mother that the child is suffering with a cough, difficulty in breathing, and that three months ago it had an attack of measles. I asked if the child broke out thoroughly, and she said it did not. We find the baby's temperature at the present time, taken in the axilla, is 103° F., and it is probable if carefully taken in the mouth or rectum it would be found a degree higher; the skin has a burning, pungent feeling to the touch, indicating a considerable degree of surface temperature.

We find that the eruption during the attack of measles was sparse and scattering. I desire to call your attention to one point in regard to the exanthemata which will apply to all. It is a very trite saying and it is understood by the laity, and they will often say to you, "Doctor, if this does not break out well, what shall we do?" The laity are impressed with the idea that the eruption should be copious in order to avoid complications. This is based upon truth. In scarlet fever and measles we are likely to have complications in inverse proportion to the development of the rash or eruption,—i.e., if the patient has a copious eruption complications are unlikely, and *vice versa*.

In the study of measles we find that the disease shows a decided predilection for the mucous surfaces; it has an affinity for the mucous membrane; it is a microbic disease, and the complications of measles will be in line of this affinity. So you may expect as complications and sequelæ involvement of such organs and parts of the body as possess a mucous lining. The bronchial tract is

always affected, and this must not be considered a complication, but a necessary part of the measles. Coryza, cough, and bronchitis precede the eruption, and it is found in such cases that the mucous surface of the bronchial tubes is the seat of measles eruption before it appears upon the external surface of the body. Sometimes just before the rash comes out you may be able to detect upon the buccal mucous membrane, sometimes over the fauces and hard palate, the eruption of measles. I have frequently been able to detect such a condition.

The next complication of measles would be a resulting bronchopneumonia from inflammation of the bronchial mucous membrane or the air-cells and capillary bronchial tubes. This is in part an extension by continuity of structure, so that while bronchitis is always present, bronchopneumonia is frequently present as a consequence of the preceding bronchitis. These are the expected complications.

The next most frequent would be involvement of the mucous lining of the intestinal tract. We have entero-colitis frequently upon decline of the eruption. There is always a tendency to irritation of the intestinal tract and diarrhoea in measles at about the time the eruption declines. Sometimes this amounts to so much that it requires especial treatment, and constitutes a complication which is exceedingly hard to manage.

Next we have inflammations of the conjunctivæ; this is a part of measles generally, but sometimes it is carried to such an extent as to require especial care and treatment. In all cases it is necessary to exercise care in protection of the eyes. The conjunctival membrane is inflamed, and this applies to the ocular as well as the palpebral conjunctiva.

Then we have the genital parts affected as a consequence of measles. We may have a persistent catarrhal discharge in the female child, constituting a vulvo-vaginitis, showing very plainly that the poison of measles expends itself on the mucous surfaces. The distribution of the poison or the micro-organisms, as the case may be, to the skin relieves the internal manifestations, and just in proportion as we have a thoroughly developed eruption the internal symptoms are held in abeyance, and the reverse of this is equally true. If we have a sparse eruption we may expect complications. This is not too far removed from measles, then, to be considered as an etiological factor in the determination of bronchopneumonia

which complicates some of these cases. You will, therefore, see the great importance of producing upon the skin a thoroughly well-developed rash. This is nature's way of getting rid of the poison, and all those means which tend to promote a rash upon the skin are legitimate in the treatment of the initial stage of measles.

Measles is longer in duration from the time of inception of the fever until the eruption makes its appearance than any of the other eruptive diseases. It takes four days of fever, during which there is constant cough, coryza, profuse lachrymation, sneezing, and all the distressing symptoms of a bad cold before development of the measles rash. The fact that it requires four instead of three days for the declaration of the rash is of considerable assistance in the diagnosis. It is the only one of the eruptive diseases in which the eruption is preceded by a fall of temperature, and this becomes characteristic. For instance, if you are called to a case in which the disturbance commences with sneezing which is persistent, lachrymation which is excessive, cough, coryza, intolerance to light, etc., I think you can safely predict measles, especially when there is a prevailing epidemic. The character of the cough preceding measles deserves especial mention; it is a croupal cough, a cough that is made within the box of the larynx, so to speak, a laryngeal cough which gives a peculiar croupal sound. This is so marked that in many instances physicians have made a diagnosis of catarrhal croup, and afterwards found that the disease was measles. This is a common mistake, though it cannot be properly called a mistake, the symptoms are in the larynx, the cough is croupal, and I have been called to many cases in which I withheld the diagnosis for a couple of days and found that I was dealing with measles. So I would have you on your guard as to the peculiar character of the cough if it is accompanied by sneezing and lachrymation. Sometimes measles produces such an intense inflammation of the larynx that a genuine membranous croup complicates the disease. It is not uncommon for measles to precede an attack of croup. Measles constitutes a very good means of preparing the field for either croup or diphtheria if these diseases prevail. The eruption, irritation, and denudation of the throat by the constant coughing affords good opportunity or resting-place for other micro-organisms.

In measles there is a drop in temperature which becomes characteristic and which occurs on the third day. We have a rise in

temperature for two days, during which time it may register  $103^{\circ}$  to  $104^{\circ}$  F., then the temperature recedes to about  $100^{\circ}$ , and the symptoms seem to be more in check, and this is just at the time preceding the rash. In other eruptive diseases the high temperature is maintained, the rash appears, and the temperature subsides afterwards. Measles acts very differently, and this is the cause of frequent mistakes in diagnosis. You may be called to see a little child suffering with what appears to be a bad cold, there is fever, difficulty in breathing, etc., and on the third day there is a marked diminution in the temperature; if you have had measles in your mind during the preceding two days, unless you will remember the points I have made in this connection, you are apt to say the child has not a case of measles, it is better, etc. Now this is just the time when the eruption will make its appearance upon the skin. It is the determination to the skin which produces the fall in temperature. This characteristic is almost invariably noticed, and I regard it as an important diagnostic feature. After the rash appears upon the skin then the temperature again goes up and remains high during the eruptive period; during this time it may remain  $103^{\circ}$  to  $104^{\circ}$ , without the development of any complications. The rash begins to decline about the seventh day of the disease. That is to say, the eruption about the face and upper part of the body begins to fade first, and later it declines from the legs and lower extremities, usually about the seventh day. For a week thereafter, when the child becomes chilled or the skin is cool, blue spots can be seen in place of the maculæ of measles. I have seen this condition last for two weeks after the disappearance of the eruption. In case of death during the eruptive stage the appearance of the corpse is peculiar, having a mottled, blue characteristic appearance. This, however, is not alone a characteristic of measles, as when death occurs during the eruptive stage of any of the exanthemata the same condition is observed.

The point in which we are especially interested is to develop the rash in such a manner as to prevent the possibility of any complications, and the question is, How are we to do this? I have seen to-day several cases of measles; an epidemic is prevailing in Louisville at the present time; it is one of the most universal epidemics, without being severe in character, that I have ever seen. I understand that out in the surrounding country the disease is prevailing just as

in the city, and it is probable that few children will escape. I regard the occurrence of such an epidemic rather fortunate; measles is not an especially dangerous disease in childhood, but it is sometimes exceedingly dangerous in adult life, for the reason that in the latter it is not usually so well cared for. I believe it is fortunate for children to have measles while they are under the parental roof and under maternal care. It is a misfortune to escape the disease in childhood and have it in adult life. Measles differs from a great many other diseases in this respect: there are some of the exanthemata which if you escape in childhood you are not likely to have in after-life, but this is not true of measles. If you escape it in childhood the immunity in after-life does not exist, and you are likely to come in contact with it and take it. More people have measles than any other of the eruptive diseases; then, again, measles does not secure the same immunity to a return or to a second attack that some of the exanthemata do. If the statements of patients can be relied upon, measles occurs with considerable frequency the second time, but I would advise you to accept such statements *cum grano salis*, for the reason that in infancy there are many innominate rashes that are called measles, and we must conclude that many similar statements grow out of the fact that people have been told they had measles when in reality they did not. During the period of eruption of the teeth there are many rashes that cannot be named which are due to reflex irritation, and sometimes the statement may be made that the child has measles. I account for many of the extremely light cases of measles in this way,—they are not cases of measles at all. The statement has been repeatedly made to me during the present epidemic, “Why, doctor, my baby had measles when it was six or eight months old.” When asked if the baby was very sick at the time, if it had a cough, sneezing, coryza, etc., the reply would be in the negative, but they said, “It had a rash which lasted for a day or two, then passed away, and the doctor said it was measles.” Such statements as these are certainly misleading; you must remember that we cannot have genuine measles without the lesions that go with it, and which have been enumerated above. However, we may have without these characteristic manifestations what is known as false measles, called by some authors German measles, rubella in contradistinction to rubeola, which is characteristic in the appearance of measles, and used to be called *morbilli*

*sine catarrhalis* before it was known to be caused by a separate microbe. This false measles, as it is called, has none of the symptoms of measles excepting the rash. There is in such cases a slight sore throat, a high temperature, and a perfect maculated rash all over the body, and these symptoms last for a period of several days, then gradually decline. It is highly contagious, propagating its own kind, which is a disease about half-way between measles and scarlet fever, and its determining microbe, whatever it is, which has not been yet isolated nor named, is evidently a hybrid, as it produces a condition partaking of the nature of two diseases, and protecting in my judgment from neither. The disease possesses some of the characteristics of measles and some of those of scarlet fever, but is distinctly different from either. Just how to account for this disease would be difficult unless we assume that the microbe of measles and the microbe of scarlet fever having become acquainted, and (like the human family sometimes do) have paid some attention to each other, or have gotten married and therefore produced a hybrid, just as we know the mule is the offspring of the horse and the ass. So it may be that the microbe of measles and the microbe of scarlet fever have married, and as a result we have a hybrid microbe which produces r  theln, or false measles. I have encountered this disease many times in families, and in my judgment it does not protect either against itself in another attack or against true measles or scarlet fever. You should always be on your guard, then, in diagnosing measles entirely from the eruption, and I may say that measles cannot be distinctly diagnosed by the eruption. You have probably read a great deal about the crescentic form of eruption in measles; you may find one or two macul   which are crescentic in shape, but they are rare, and when present they may be characteristic, yet their absence proves nothing. Remember that you cannot have a genuine case of measles without the catarrhal symptoms which I have mentioned, and when you are called to see a child having a rash without such catarrhal symptoms, you had better call it false measles.

To return to the treatment, and the importance of bringing about a thoroughly developed rash. As I have said before, the question is, How shall this be accomplished? Delay of the rash in measles is always unfortunate. Before coming into the lecture-room I had just returned from a visit where the entire family is down with the measles; one child was taken sick with measles eight days

ago, and the statement of the father to me was that it broke out very poorly. There was some cough, also some lachrymation. The rash broke out very sparsely, and in a few days the child was able to be up about the room. Meantime I was called in by the family. I found the child with a temperature of  $104^{\circ}$  F., with rapid breathing, severe bronchial symptoms, and with the characteristic cough of measles. I stated that I believed it was a reinfection, or that the measles had not been developed properly in this case, and that I would make an attempt to develop the rash and thus relieve the internal symptoms. The plan I followed was to take a tablespoonful of mustard and place it in a quart of hot water, then wring a thick towel out of this mixture and wrap it around the child's body. In this way I was able to develop the rash very promptly. I saw the child this morning, and it is thoroughly broken out from head to foot with measles eruption. I gave internally such medicine as would determine the disease to the skin.

Here is undoubtedly a case of suppressed rash of measles with a very sick child. With the breaking out of the rash the bronchial symptoms will disappear, the temperature will decline, and the child will feel much more comfortable. As far as internal medication goes I believe that iodide of potassium is the best agent we have for determining the rash to the skin, and I have used it for many years with marked success. There is another agent, however, which I really prefer for this purpose,—viz., syrup of hydriodic acid. My favorite prescription for such cases in the initial stage is perhaps worth remembering; it is as follows:

R Syrup of hydriodic acid,  
Syrup of tolu,  
Syrup of Dover's powder, aa  $\mathfrak{z}$ i. M.  
Sig.—A teaspoonful every two hours.

This makes a very pleasant mixture. You will at once see the value of this preparation in allaying the cough, quieting the irritation of the throat, and it is also a sudorific which aids in determining the rash to the skin. The child should be put in a warm, darkened room, which must be kept at an even temperature, about  $70^{\circ}$  F., and if this plan is followed you will seldom fail to develop the rash promptly and thoroughly. I have found this to be such an excellent prescription that I never change it except to vary the amount of Dover's powder occasionally to meet the indications in a given case.



This mixture affords relief to the bronchitis at the same time. In delayed cases, such as I have referred to, it may be necessary to resort to the plan of enveloping the child in towels wrung out of hot mustard water, as described, but ordinarily this will not be called for. The child should be kept warm, but not over-heated, and the room kept at an even temperature. You will find that the bronchitis will vary in a few hours by a change in the temperature of the room. When the child has a cough that sounds loose, not attended by dryness of the mucous surface, there is no danger; but if the temperature of the room is materially lowered, even for a short time, you will find that the character of the cough changes correspondingly; it becomes hard and dry and the difficulty in breathing is greatly increased. Therefore, in the treatment of bronchitis, whether as a complication of measles or otherwise, the temperature of the room should be kept at about 70° F. night and day; and it is of wonderful benefit to have the atmosphere of the room slightly moist, which may be accomplished by having a tea-kettle or other vessel containing water over the fire in cold weather, or heated and brought into the room in warm weather, thus filling the room with steam and keeping the atmosphere moist. Another good plan is to partially fill a bowl or tub with water and then place in it bricks which have been heated as hot as possible, thus filling the room with vapor.

As regards the use of diluents internally to determine to the skin the rash of measles. The laity are greatly imbued with the idea that hot drinks and tisanes are beneficial because they make perspiration, and there is some truth in this. Let them drink hot teas, hot lemonades, stimulants, such as whiskey, etc. It is true you may make a person sweat by giving him hot drinks of any kind, even hot water, but the sweating will not be any more determined to the skin than if you give him cold drinks, and hot drinks are certainly less pleasant. You may sweat in the summer-time by drinking hot tea or hot coffee, but you will suffer a great deal more inconvenience than you would by drinking ice-water, which would cause just as free perspiration. Therefore I say, while it will do no harm to allow a child with measles to have hot drinks, still I think the better plan is to insist upon their drinking plenty of cold water, which determines to the skin as well as to the kidneys and will be productive of good results. You will get a very good idea of the use of hot water and other hot drinks in the treatment of measles in your private practice. Some mothers will hold up their hands and say

to you, "Why, doctor, you would not have me give this child cold water to drink while it has measles, would you?" To this your reply should be, "Yes, and give plenty of it; the child is fairly burning up with fever and does not need hot drinks." But you will find that the laity are opposed to giving cold water, and will not do so unless you enforce it.

In the case before us we have had a bronchitis as a result of measles; it has now reached the capillary bronchial tubes by continuity of structure, and we have what is known as catarrhal pneumonia, which is the characteristic pneumonia following measles. It is a tedious form of pneumonia to treat. Compared with croupous pneumonia it is not so frightful in its early manifestations, but its course is usually protracted, and it is likely to return, subject to ameliorations and exacerbations, until the child is practically worn out, yet it is generally considered the less dangerous disease of the two. For my own part I would prefer to treat a case of regular croupous pneumonia, which is a microbic disease due to a specific organism, and has a limit as to its duration, than to treat a case of catarrhal pneumonia. Croupous pneumonia undergoes resolution in from five to seven days; bronchopneumonia may last one week, two weeks, or three months, in which time the child may present symptoms of being better, then worse. It is really not pneumonia at all, it is capillary bronchitis, the pneumonic symptoms being simply due to collapse of certain portions of the lung, consolidation from filling of the lung with inspissated mucus, etc. In an examination to-day you may find a certain portion of the lung over which you can get the symptoms of dulness, to-morrow that part of the lung may be clear, and some other part is dull, so the process wanders over the lung. In infants the disease is exceedingly dangerous. The child before us is old enough to withstand the disease, I think, and the prognosis is favorable, but in younger infants the prognosis of bronchopneumonia is exceedingly grave because of their inability to get rid of the inspissated mucus. The mode of death in these two kinds of pneumonia is worthy of our attention. In bronchopneumonia it is incumbent upon you to carefully watch the respiration and respiratory changes which take place, for upon proper respiration depends the oxygenation of the blood. Respiration is to be watched, for death comes in these cases, if it comes at all, through interference with the proper oxygenation of the blood, evidenced by blueness of the lips, blueness of the extremi-

ties, a marble-like condition of the cheeks,—all these are symptoms which we do not like to see in catarrhal pneumonia. In the other form of pneumonia we leave the lung out of consideration so far as watching the patient is concerned, knowing that the pneumonic process will go on to resolution even without treatment. In fact, in croupous pneumonia, outside of watching the heart, it may be said that the disease gets along just as well without treatment as with it. It is not so with catarrhal pneumonia. In croupous pneumonia the danger comes from the heart from the amount of the area of consolidation in the lung preventing the blood circulating around the circuit, and death comes from over-distention of the right ventricle. Therefore it is well to remember that death occurs from failure of the heart in croupous pneumonia, and from failure of the lungs in catarrhal pneumonia. This teaches you to watch two different things in the two diseases. Pay all your attention to interference with the respiration in catarrhal pneumonia; so long as the cheeks are red, so long as there is no blueness, so long as the child can cough freely, there is but little danger. High temperature is not alarming unless attended with pale skin and a great deal of dyspnoea. Paleness of the skin, contraction of the arterioles, with high temperature, is quite a different thing.

The question is what do we expect to do in a case of catarrhal pneumonia by treatment? We cannot cut it short; we might have prevented it in this case if we had treated the measles, but it is too late now. What are the indications as to medication? is the question the doctor asks himself when he visits one of these little patients. The first thing to do is to lessen the viscosity of the mucus, which is poured out in such abundant quantities as to fill the air-cells. Therefore such agents as have a tendency to liquefy the mucus are beneficial. We have no agents equal to some of the potassium salts for this purpose. We want to render the sputum less viscid so it may be expectorated, then we want to stimulate expectoration. In doing this we shall accomplish all that is desired, and these are the cases in which the so-called fever mixture works wonders; it is the only thing that is required, with one exception—plenty of water.

R Potassii acetatis, ℥ii;  
Spiritus Mindereri,  
Aquæ camphoræ, aa ℥iii. M.

Sig.—A teaspoonful every hour for child; tablespoonful every two hours for adult.

Now, if you were to treat this little patient for bronchitis, or bronchopneumonia, with simple medication without instructions to give the child plenty of water, you would fail to produce the desired result. If I were asked the question what is the best expectorant in the whole materia medica, I would have to say water. You should urge the use of water. A child when suffering with fever needs very little urging, it will drink a half-cupful every hour, and if it does this the cough will become loose, expectoration easy, and all the symptoms will improve.

As regards the fever mixture: it contains acetate of potassium, ether, and camphor water; the dose for infants is a teaspoonful every hour, for older children a dessertspoonful, adults a tablespoonful. It is wonderful the improvement that will be brought about in a few hours by the administration of this simple medicine. All the distressing symptoms disappear usually in three to four hours.

The question has been asked as regards local applications in this character of pneumonia. In bronchopneumonia if we were to make local applications what do we expect to gain thereby? I believe that the inunction of liniments, etc., in this disease is of very little value. A very good plan is to envelop the thorax with absorbent cotton covered with oiled silk so as to make an impervious covering, letting this remain until improvement takes place, but I would not advise the use of liniments or washes.

In regard to poultices: such treatment in bronchopneumonia would be most injudicious. To apply a poultice of flaxseed or other material, as is sometimes done, around the chest for the child to elevate every time it breathes, requiring that much more power in the act of respiration, cannot be considered good treatment. I would not raise the same objection to a poultice in a case of croupous pneumonia where the lung was undergoing resolution in which moisture might be beneficial.

In the case before us we will prescribe the fever mixture, and instruct that the child be cared for as indicated in the foregoing, that it be encouraged to drink plenty of cold water, and we believe a complete cure will result, although, as I have already stated, in some cases the disease will undergo periods of amelioration and exacerbation for a considerable length of time. My experience, however, is that since I adopted the use of the treatment outlined in this lecture I have had less trouble than when other methods were employed.

## A CASE OF SPLENIC LEUKÆMIA, WITH OBSERVATIONS UPON THE CHARACTER OF THE BLOOD IN THIS DISEASE.

CLINICAL LECTURE DELIVERED IN THE GLASGOW ROYAL INFIRMARY.

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GENTLEMEN,—Leucocythæmia (Hughes Bennett) or leukæmia (Virchow) is a disease of sufficiently rare occurrence to merit a careful study and description of each case as it arises. The essential features of the affection have been well known to the profession since 1845, when they were almost simultaneously but quite independently described by Hughes Bennett, of Edinburgh, and Virchow. The outstanding phenomenon of the disease is an enormous increase of the white blood-corpuscles, so that in severe cases they may be practically equal in number to the red disks. Recent researches on the lines inaugurated by Ehrlich have led to a much more perfect knowledge of the minute changes occurring in the blood, and have enabled pathologists to recognize well differentiated varieties of leukæmia. In all varieties of true leukæmia the increase in the number of the white blood-corpuscles is an essential factor, and hence in our present study any reference to so-called pseudoleukæmia may be omitted. Two varieties of leukæmia are so sharply demarcated by the microscopical characters of the blood that they may practically be regarded as separate and independent affections: They are: 1. Splenic leukæmia, which has also received the following designations: splenomyelogenous leukæmia, lienomyelogenous leukæmia, myelæmia, leukæmia lienalis, and splenomedullary leukæmia (Muir). 2. Lymphatic leukæmia, to which the name lymphæmia has also been applied.

These varieties constitute two well-marked types of the disease, but Rieder in his atlas of the blood has described and illustrated

an intermediate form, to which he has given the name of "gemischte leukæmia." Further discussion of the differential characters of these varieties of leukæmia may be left over until I have described the symptoms of the patient in Ward 7, who is suffering from the splenomedullary form, and referred more briefly to other cases of the disease which have from time to time come under my observation.

Thorwald T., aged eighteen, a Norwegian sailor, was admitted to the hospital on the 17th of January, 1898, complaining of diarrhoea of six weeks' duration, and of swelling of the feet and ankles since the preceding day. The captain of his ship believed that he was suffering from beriberi, and sent him to the Infirmary with an intimation to that effect.

Inquiry elicited the following information as to his previous history: As a child he had suffered from no illness except thread-worms. Two years before his admission, on leaving Mobile, a seaport of Alabama on the Gulf of Mexico, he developed on board ship a sharp attack of malarial fever, which lasted for a month, confining him entirely to his bunk for a period of ten days. Six months after this he had another attack, which lasted only a few days. A year later while in the West Indies he passed through an attack of yellow fever, and shortly before this, while his ship was at one of the ports of Brazil, he spent ten days in a hospital on account of a rheumatic affection of the left hip. A careful inquiry as to the ordinary symptoms of beriberi yielded no evidence of his ever having suffered from that disease.

With the exceptions just mentioned he remained well and able for his work until six weeks ago, when, on his way home from Jamaica, he began to suffer from diarrhoea. In spite of this and of the circumstance that he was losing flesh he remained at his work until Sunday, the 16th of January, when he had become very weak and his feet had commenced to swell. On this account he was sent to the hospital on the 17th.

His family history is unimportant. His parents died while he was very young. He has one brother alive and well. Two sisters died in infancy, and a brother died at the age of eight years.

In proceeding to demonstrate to you the clinical phenomena of this patient's case I may confine myself to the salient features of his disease, referring you for information as to the state of his

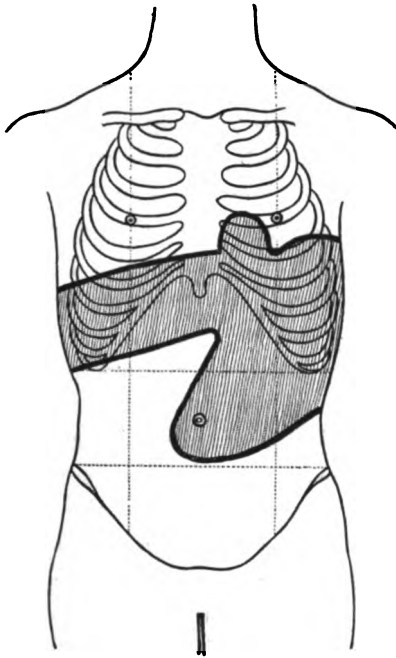
organs in general to the detailed report in the ward journal. As you see, he is a well-built and fairly nourished young man. His complexion is sallow, on the whole rather suggestive of malarial cachexia, but certainly not at all indicative of pronounced anæmia. The tongue is clean; the pulse is full and soft, and regular in force and rhythm, numbering about 80 per minute. There is no paralysis; sensation is everywhere perfect; the knee-jerks are normal; there is no ankle-clonus, and œdema has entirely disappeared. Without going into the details of the physical examination it may be stated generally that his heart and lungs present healthy characters; his urine contains neither sugar nor albumen; his temperature is practically normal; his appetite has been fairly good, and his diarrhœa is now almost well.

On inspection as he lies in bed you observe a distinct uniform and practically symmetrical enlargement of the abdomen. Palpation at once reveals a large, solid mass filling the left side of the abdominal cavity, which extends from the left axillary region to the hypogastric region, and which we have no difficulty in at once recognizing as a greatly enlarged spleen. Its surface is smooth, and in the epigastric region the splenic notch can be easily felt. The percussion over the enlarged organ is dull, and the greatest oblique diameter, from where the line of dulness cuts the sixth rib in the mid-axillary line to the lowest point in the hypogastric region at which its lower end can be felt, is sixteen inches. In the anterior axillary line the greatest vertical diameter is twelve inches. The organ has been increasing in size during the two months he has been under observation, and the diagrams I now show will enable you to compare its size to-day with that made out by the same methods on the 26th of January last. The hepatic area is not enlarged, nor is the cardiac, though the latter is now perhaps slightly higher than normal, the heart having been pushed up a little by the greatly enlarged spleen. The lymphatic glands in the axilla are unduly palpable, and those of the groin are distinctly though slightly enlarged.

The possibility of the enlarged spleen being a malarial manifestation is at once removed by an examination of the blood, specimens of which I have placed under the microscope for your inspection. You will immediately perceive the enormous number of white blood-corpuscles, which are present in the proportion of one white to

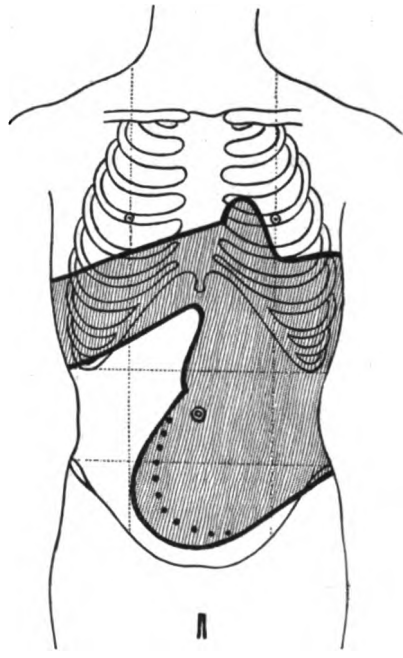
six red corpuscles. The red corpuscles number two millions to the cubic millimetre of blood, a decided anæmia, and the hæmoglobin is equal to thirty-five per cent. No melanin granules have ever been seen.

FIG. 1.



Shows outline of splenic enlargement as observed on January 25, 1898.

FIG. 2.



Outline as observed March 18, 1898. The dotted line indicates the area of percussion dulness; palpation carrying the lower margin of the spleen one and one-quarter inches lower down.

To my clinical assistant, Dr. T. K. Monro, I am indebted for the following report of his ophthalmoscopic examination: "*Right eye*: both arteries and veins very tortuous; calibre of arteries not distinctly altered; fundus otherwise normal. *Left eye*: arteries somewhat tortuous, veins broad and tortuous; small flame-shaped hemorrhage below the disk; otherwise fundus normal."

The history which I have read to you and the clinical facts which I have demonstrated leave no doubt that we are here dealing with a typically characteristic case of splenic leukæmia. Before passing on to describe very briefly the other cases of which I have notes and to speak more in detail of the condition of the blood, I may state that during his residence here, notwithstanding the increase in the size of



the spleen, the general condition of the patient has improved. While at the convalescent home a week or two ago he contracted a bronchial catarrh and was slightly feverish for a day or two, but this has now passed off. His blood has not altered in any appreciable way, although a recent count showed the white blood-corpuscles now to be in the proportion of 1 to 9. He is now on treatment by five-minim doses of Fowler's solution, and by inunction over the spleen of red oxide of mercury ointment.

Including the case I have just demonstrated to you I have personally seen and examined five cases of leukæmia, three in the wards as a clinical physician, and two in the dead-house as a pathologist. Four of the five cases were of the splenomyelogenous and one of the lymphatic type of the disease. Of the three clinical cases I need say nothing more than that all of them more or less closely resembled the case you have seen this morning, both as regards the characters of the spleen and the blood. With regard to the post-mortem cases I may enter a little more into detail.

One of them was very characteristically of the splenomyelogenous type. The patient, a man aged twenty-six, died in the wards of my colleague, Dr. Alexander Robertson, and I made the post-mortem examination upon the 29th of June, 1890. At my request Dr. T. K. Monro, then working in my laboratory, undertook the histological examination of the case, and you will find a careful account of his observations in the *Glasgow Medical Journal* for May, 1892, at page 353. I may say that the chief lesions were found in the spleen and in the bone-marrow. The spleen weighed eight pounds two and one-half ounces, and its dimensions were, length thirteen and one-half inches, breadth eight inches, and anteroposterior diameter five inches. One-half of the organ, which I now show you, has been preserved in our museum, and you can appreciate the enormous enlargement which had taken place. This colored drawing, executed by my sister, Mrs. J. H. Connell, gives a faithful picture of the appearances in the fresh state. The cut surface had an appearance somewhat like that of red marble, a mixture of red and white areas of various shapes and sizes. The large yellow caseous-looking patches of irregular shape, resembling very closely embolic infarctions, are areas of necrosis. Dr. Monro found that, while the Malpighian bodies were scanty, a great new formation of lymphatic tissue had taken place throughout the pulp. Here and

there the sinuses of the pulp were filled with leucocytes in larger numbers than the red blood-corpuscles. The leucocytes in the sinuses were often very large, and sometimes contained pigment. The necrotic and the living portions of the splenic tissue alternated with one another in an irregular fashion. The fibrous tissue of the pulp was infiltrated in many places with pigment of a brownish-red color. I have thus somewhat fully summarized the detailed description of the spleen in this case because I think you may take it that the spleen of our patient is likely to be in much the same condition. The marrow of the shaft of the femur was replaced by lymphatic tissue, which, however, had not in any way destroyed the bone. All the capillaries of the organs examined were found to be crowded with white blood-corpuscles, but, except in the spleen and the bone-marrow, there was very little new-formation of lymphatic tissue. I may add also that the patient had suffered from malaria.

The other post-mortem case of leukæmia occurred many years ago, when I was assistant to Professor Joseph Coats at the Western Infirmary. At that time the distinction between the different varieties of leucocytes had not been definitely formulated, but, as I made and frequently examined many sections of the tissues in this case, I can now see that it was a very characteristic example of the lymphatic or lymphæmic variety of leukæmia. New formations of lymphoid tissue were very abundantly present in most of the tissues and organs of the body, a circumstance in striking contrast with the histology of the splenic case examined by Dr. Monro. In the kidneys, in the liver, and in the pericardium, as well as in the spleen and the bone-marrow, typical new-growths of lymphoid tissue could easily be demonstrated, while the dense overcrowding of the capillaries with leucocytes could not be so easily made out. The lymphatic glands in this case were also enlarged, apparently from a hypertrophy of their tissue. This is the class of case which is likely to give rise to the discussion of the possible relationship of lymphatic leukæmia to Hodgkin's disease.

Having thus briefly referred to the anatomical peculiarities of the two chief forms of leukæmia as illustrated by cases occurring within my own experience, I desire now to describe more in detail the characters of the blood in our patient's case. The specimens under the microscopes have been very carefully and beautifully stained by my assistant, Dr. Walter K. Hunter, who has devoted

much attention to the clinical examination of the blood. In these specimens you will easily make out all the characteristic corpuscles met with in splenic leukæmia. You will observe:

1. That the red blood-corpuscles are on the whole normal in size and shape. Here and there nucleated red blood-corpuscles are to be seen.

2. That eosinophile corpuscles are fairly numerous, the red eosin-stained granules of the protoplasm and the pale blue nucleus imparting to these cells a striking and beautiful appearance. Sometimes you will notice that the corpuscle has burst, scattering the eosinophile granules over a moderately wide area of the field.

3. That polynuclear leucocytes with their nodulated irregularly-shaped nuclei of a deep blue color and their pale, slightly pink protoplasm are numerous.

4. That large mononuclear leucocytes, often almost entirely filled by a pale blue nucleus, the so-called myelogenous or marrow-cells, are the most numerous of all.

5. That lymphocytes, the small mononuclear leucocytes, are almost absent, only one or two being very occasionally observed in a microscopical field.

It is not my intention to discuss the staining reactions of the different forms of leucocytes, a subject of great importance and of which our knowledge has been greatly increased in recent years, but I shall content myself by informing you that Dr. W. K. Hunter has embodied his views in a paper, communicated to the Glasgow Medico-chirurgical Society, which I hope you will soon be able to study in its published form in the *Glasgow Medical Journal*. It is sufficient at present to say that the features of the blood in our case correspond in almost every detail with those described by other observers as characteristic of splenic leukæmia. From the microscopical appearances which you have just made out I think it would be possible without other help to diagnose the disease. Let me now very briefly indicate to you the characters of the blood in the lymphatic variety of the disease. Lymphocytes are enormously increased in number. Polynuclear leucocytes are not more numerous than usual, and eosinophile corpuscles are very scanty. Myelogenous or marrow-cells are not met with, and nucleated red blood-corpuscles, except when anæmia is a marked feature, are not present. You will see, then, that by a careful examination of the blood alone

it is possible to distinguish the two varieties of leukæmia. The mere presence of enlarged lymphatic glands would be insufficient, for, as you have to-day seen, the glands may be swollen in characteristic cases of the splenomyelogenous variety.

There are, however, some other points connected with the blood in our patient's case to which I desire to direct your attention. In view of the clear clinical history of malaria a careful search has been made by Dr. Hunter for the plasmodium malaris, but entirely without success. This, as I shall point out later, is a point of some importance. I directed your attention a little while ago to the pigment found in the spleen in Dr. Robertson's case. Melanin granules are familiar objects in the histology of ague, as I have seen on several occasions, and it is possible that the somewhat extensive prevalence of pigmentation in the spleen in Dr. Robertson's case may have been due to the malaria. In our case, however, no pigment granules were found in the blood.

We also carefully examined the blood for other micro-organisms. In specimens of fresh blood mounted by Dr. W. K. Hunter and by my resident assistant, Dr. William Martin, I saw on several occasions, with the one-twelfth oil immersion lens of Leitz, actively moving micrococci. Every precaution was taken to prevent contamination of the drop of blood by sterilizing the finger, the slide, and the instruments, and it might fairly be presumed that we had succeeded in preventing the accidental introduction of germs. The movements of the minute round bodies were not Brownian, upon which we were all agreed. Many attempts were made to cultivate germs from the blood, but without success. All the tubes remained sterile after lengthened periods in the incubator, a proof, I think, that germs had not been introduced from the outside. Dr. Hunter, however, who has succeeded in cultivating germs from the blood in cases of beriberi, informs me that the cultivation of germs from the blood is a matter of great difficulty, so that our want of success need not discourage us unduly in reference to future attempts. Meanwhile, with the exception of the observation of motile micrococci in the freshly-drawn specimen, our efforts to demonstrate organisms in the blood of leukæmia have been without result.

I shall conclude this lecture by making a few general remarks upon the etiology, symptoms, and course of the disease we have been studying to-day.

As regards etiology it must be admitted that we know very little, and the only point under this heading which I desire to discuss is the relationship of leukæmia to malaria. That there is a relationship may be admitted. In two at least of my five cases there was a clear clinical history of antecedent attacks of ague. On this point, in the paper before referred to, Dr. T. K. Monro writes as follows: "A study even of the earliest recorded cases of leukæmia—as, for instance, those collected in Bennett's work on the subject—suggests a connection between this disease and intermittent fever. Virchow remarked on this as early as 1852 (*Archiv* for 1853, vol. v., p. 95). In a discussion at the Pathological Society of London in the year 1878, Gull, Gowers, and Goodhart all expressed their belief in such a relationship. Gowers stated that there was a history of ague, or of residence in an ague district, in twenty-five per cent. of the cases of splenic leukæmia which he had collected (*Lancet*, 1878, vol. i., pp. 460 and 495)." Notwithstanding the frequency with which malaria forms an event in the clinical history of leukæmia, its etiological significance has been doubted by certain writers, and I may read to you Osler's opinion as expressed in Pepper's "Text-Book of Medicine," vol. ii., p. 215: "That malaria and syphilis bear any etiological relation to leukæmia is scarcely probable, although in one hundred and fifty cases analyzed by Gowers thirty had a malarial history, and over one-third of my cases had previously suffered from malarial invasion." I must say, however, that, on theoretical considerations, the possibility of leukæmia resulting from a miasmatic poison is a view which recommends itself to me.

Among the prominent initial symptoms of our patient's case you will recollect that diarrhœa was one. Gastro-intestinal symptoms are not uncommon as early manifestations of the disease, and it has been suggested that this circumstance may not be without etiological significance. It is possible that the affected intestine may be the "gateway of entrance (infection-atrium) for the leukæmic virus" (Osler). In favor of such a view as to the significance of initial diarrhœa or dysentery I may refer you to the frequency with which various forms of anæmia and toxæmic jaundice are associated with structural lesions or functional disturbances of the alimentary canal.

You will all remember a clinical lecture which I delivered during the present session on a case of toxæmic (non-obstructive?) jaundice, which during life we thought might possibly prove to be

an illustration of acute yellow atrophy of the liver. On post-mortem examination no obvious obstruction to account for the intense jaundice could be found, until a microscopical examination of the liver had been made, when the capillary bile-ducts were found to be obstructed by inspissated bile. The patient had suffered from an initial diarrhœa, and the colon was beset by numerous small superficial circular ulcers, from which poisonous matters might easily have been absorbed.

Dr. Monro discovered in the retina of the left eye of our patient a flame-shaped hemorrhage. This leads me to say that hemorrhages—epistaxis, hæmatemesis, melæna—are not at all infrequent symptoms of leukæmia. In this connection the practical point I desire to impress upon you is that you should never undertake even the most simple cutting operation on a patient suffering from splenic leukæmia without recollecting, and preparing yourself for, the circumstance that you may have great difficulty in arresting the hemorrhage. In one of my cases I had unfortunately an experience of the truth of this observation. The man developed a glandular abscess in the submaxillary region, and I requested my surgical colleague, Mr. Henry E. Clark, to open it for me. This he did, evacuating a large quantity of pus. Soon after, however, the abscess cavity was filled with blood, and it was only with the greatest difficulty and after the patient had been reduced to death's door that we succeeded in arresting the hemorrhage. Please remember this experience of mine if you are ever called upon to deal with a similar case.

With regard to the course of splenic leukæmia I have to point out to you that the disease progresses, as a rule, slowly. During the two months that our patient has been under observation his condition has undergone but little change, with the exception that the spleen is now much larger than it was at the time of admission. Osler points out that "the majority of cases terminate fatally in two or three years," although during that time there may be intervals during which the patient enjoys fairly good health. Lymphatic leukæmia, on the other hand, is said to run a much more rapid course, and in some very acute cases death may ensue in a few days or weeks.

As regards the treatment of the disease, it must be admitted that on the whole we have been able to accomplish very little. In addi-

tion to regimen and hygienic measures, I think you will obtain the most beneficial results from the cautious and persistent use of Fowler's solution, in doses of about five minims administered three times a day, and stopped when physiological effects show themselves. There could be no harm in trying the effect of the administration of bone-marrow or splenic extract, numerous preparations of which are now in the market, but of these I have had no special experience in the treatment of leukæmia. Above all, do nothing which in any way is likely to interfere with the patient's general health or to upset his digestion.

## ON THE RELATION BETWEEN HEART-DISEASE, PREGNANCY, AND CONFINEMENT.

CLINICAL LECTURE DELIVERED AT THE BAUDELLOCQUE MATERNITY.

BY A. PINARD, M.D.,

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GENTLEMEN,—The history of two women who have been confined in our wards during the last few days will give me a good opportunity to discuss for your benefit one of the most interesting and important questions in obstetrics,—namely, the bearing of organic heart-trouble on pregnancy and confinement, and the influence of the puerperal condition on heart-diseases.

It will, I think, be well for me to begin by giving you the details of these two cases, as they are *facts*; a fact is always of greater value than a statement or theory, and it is by facts that we should always be guided.

Recently a woman was transferred to our maternity wards from a general hospital. In her personal history the most noticeable features are two attacks of acute, articular rheumatism that occurred at the age of ten and fourteen years, the second attack having lasted six weeks. This woman was naturally not cognizant of medical opinions on this question, so she married later on, and had children. She was successively confined in 1882, 1884, 1886, 1888, 1890, 1892, and 1895,—seven times; for her eighth confinement, the one before the present one, she trusted herself to our care with the following result: she came to the ward at 6 A.M., and her child was born four hours later. It was at once seen that she had a cardiac lesion; there was œdema of the lower extremities and of the abdominal wall, and a systolic murmur at the apex.

Five days after her confinement dyspnœa began to manifest itself, for which inhalations of oxygen and injections of ether and caffeine were administered; but her condition did not become normal until she had taken some chloralose, first 0.10 gramme, or 1.5



grains, then 0.20 gramme, or 3 grains. The latter dose produced some disorder in her vision, and was reduced to 0.10 gramme, or 1.5 grains, again. She was at the same time put on a milk diet, which produced free diuresis. On the 30th she left us in excellent condition.

This was, therefore, an instance of a woman with a valvular lesion who went through her pregnancy in an almost normal fashion, the only pathological element being a slight dropsical condition with distressed breathing.

Not long afterwards she became pregnant for the ninth time. We found that in the ward she had just left she had suffered from dyspnœa, œdema, and cyanosis, that auscultation had revealed subcrepitant râles with tumultuous heart action, brought on by an acute infectious disorder, acute asystolia being apprehended. Dry cups were prescribed, she was given a preparation of digitalis, and in a few days' time nothing was left but a few dry, sonorous and crepitant râles.

As her general condition was fairly satisfactory we prescribed milk diet, absolute rest in a horizontal position, inhalations of oxygen, and chloralose in doses of 0.10 gramme, or 1.5 grains.

Four days later she was brought to the labor-room at 3 A.M. The dilatation had reached the size of the palm of one's hand, and in two and a half hours she was delivered. Immediately afterwards she was seized with dyspnœa and became semi-unconscious, with a pulse that could no longer be counted. Oxygen inhalations were given, and injections of ether and caffeine, after which her pulse gradually improved and numbered 88 to the minute. To-day she is again in very good condition.

To summarize: this woman's cardiac lesion was due to her attacks of inflammatory rheumatism. She had seven normal pregnancies, and nursed these seven infants without ill effects. It was only during the eighth confinement that œdema and respiratory distress manifested themselves, becoming especially marked a few days after delivery. At her ninth confinement serious gravidocardiac symptoms appeared (due to influenza, or overwork, we are not certain which), characterized by dyspnœa, œdema of the lower limbs, and disturbance of the cardiopulmonary circulation. A very ingenious and attractive theory of the latter symptom has been advanced by Peter, who claims that the sibilant râles are due to acute

congestion, which may in some cases go so far as hæmoptysis and suffocating catarrh. Absolute rest, milk diet, and maceration of digitalis were employed after the confinement, and now, as you know, the woman is in excellent condition.

Another woman in whom the diagnosis of heart-disease had been made was recently confined. Auscultation showed the usual signs of mitral stenosis and insufficiency, and we ascertained that at the age of eleven she had been nearly a year in a hospital with inflammatory rheumatism.

Now, this woman has been confined nine times since 1881, her recent confinement in our wards being her tenth! She came to the labor-room at 8 A.M.,—and examination showed left anterior position of the occiput,—and at 10.35 was delivered. There was no hemorrhage, and to-day our patient is in a normal condition.

The history of these two women will probably surprise you, as it is not in accordance with what you have read in your treatises on the relation between valvular heart-troubles and child-bearing. For this reason I shall discuss briefly the following three points:

1. Can pregnancy in itself give rise to heart-disease?

Some obstetricians have written on this point and have stated that such can be the case. The question has been raised whether the heart undergoes hypertrophy through pregnancy, and one writer who made a special study of the matter answered in the affirmative. It can be said that the greater number of French obstetricians hold that pregnancy gives rise to hypertrophy localized to the left ventricle. Some discordant voices have, however, been raised. The objection was made to the author just cited that in his cases the heart may have been diseased, and he himself finally admitted that this hypertrophy is observed in patients with eclampsia.

It seems proven that in women who die during pregnancy, not from a wasting disease but by accident, there is no cardiac hypertrophy; I am consequently very much surprised that the opinion so frequently met with, that hypertrophy of the heart in pregnancy is generally admitted, should have been written by an obstetrician. That is not my view, and I have no hesitation in saying that there is no heart-disease that can be caused by pregnancy.

2. Can pregnancy give rise to endocarditis?

This also has been claimed, and two forms have been described, one acute and the other chronic. Here again I must beg to differ,

and to say that neither form exists. Six cases of this nature have been reported to my knowledge. One of them occurred in a woman who was syphilitic and albuminuric; in her case, at any rate, it can hardly be claimed that pregnancy was the origin of endocarditis, as she was already ill before she ever became pregnant!

When the heart is not already diseased before conception, pregnancy can do it no harm; it can cause no disorder either of the peri-, endo-, or myocardium.

This once said, what is the influence of the puerperal condition on heart-disease? A vast amount has been written on this question during the last twenty years, but I am obliged to tell you that the subject is far from being elucidated. To begin with, physicians frequently disagree in their diagnosis of the variety of heart-disease. It once happened to me to meet three heart specialists in a case of this order, and to hear each one make a different diagnosis! One thought it was purely mitral stenosis; the second, stenosis with insufficiency; the third, dilatation of the right ventricle. When physicians cannot agree obstetricians can also differ.

Observe cases for yourselves, and compare the results of your observation with what has been written; you will find that it has no basis of substantial facts.

This is what you will constantly see in our wards: whatever the form of heart-disease may be, if the kidney is not affected compensation occurs and child-bearing can go through all its phases without incident; but if the kidneys are affected, and, *a fortiori*, if the myocardium is, you will see the gravido-cardiac symptoms appear, which, if not promptly and carefully treated, may be so severe as to cause death, although this is unusual.

Such in general terms is the influence of child-bearing on heart-disease; but since we rarely have any means of knowing beforehand whether the kidney will stand the strain, we cannot tell if the disorder will be compensated. Consequently it is impossible for us to decide whether this or that woman with heart-disease can bear children with safety.

3. What is the influence of heart-disease on the course of pregnancy?

In patients with heart-trouble the courses are irregular, and premature confinements more frequent than under ordinary circumstances. Let me remind you, in passing, that the placenta in cardiac trouble is whitish.

One curious point about these cases is that when a woman with heart-disease is delivered prematurely, on a second or third similar occasion the child will come still more prematurely. A case has been published of a woman with congenital cyanosis who illustrated this point in a striking manner.

Another curious fact, that must never be lost sight of, is that with these women during labor symptoms of dyspnoea and asystolia may appear very suddenly and end rapidly in death. This is particularly likely to happen in cardiac cases where the vertebral column is deformed, or where there is pleurisy, pericarditis, or hydramnios. But in these cases you have to deal with a special category of patients who cannot be classed with cases of normal pregnancy. They can also be suddenly seized with capillary bronchitis.

As regards hemorrhage during delivery in this class of patients, it is quite unusual, unless at the same time the liver is affected.

Now let me say a few words in closing concerning the prognosis and treatment in pregnancy complicated with cardiac disorder.

You have all heard what is customarily said to a woman with heart-trouble who consults her physician to know whether she ought to marry or not. She is advised not to marry at all. If she is already married, not to become pregnant. If she is pregnant, not to nurse the child. If you will take my advice you will not believe this dictum, but will be guided by your observation of cases. You can see a number of women with cardiac disorders come to our wards and be confined without any mishaps whatever, and I have just told you of a woman with congenital cyanosis who went safely through the ordeal and was none the worse for it.

I think, therefore, that we have not the right to be so absolute and to forbid women with cardiac disorder from marrying at all. Your prognosis should be based on the more or less good condition of the kidneys; you must always be very guarded if your patient has trouble with her vertebral column, or if there is any dropsy of the amnion.

The treatment to be advised is extremely simple. You must forbid all over-exertion, however slight. When a woman with a valvular lesion becomes pregnant, rest is an absolute necessity. At the same time you must watch with the greatest care the functions of the kidneys and intestines. Jaccoud's suggestion is that from the fifth month on the patient should be put on a milk diet, two

litres, or two quarts, a day, and should take digitalis in infusion or maceration.

One of the most troublesome incidents in such cases is that some of these patients find it impossible to sleep properly at night; the best remedy I have been able to find so far for this condition is chloralose, 0.10 gramme, or 1.5 grains, to 0.20 gramme, or 3 grains.

When, however, you have the misfortune to be called in to attend a woman with serious gravido-cardiac symptoms, the two main indications for saving the woman are, to empty the uterus as rapidly as possible, and to bleed; these two means form the basis of the treatment, and you must not hesitate to employ them. Venesection is specially efficacious, indeed imperative, when there is acute œdema of the lungs.

## SPRUE: ITS CAUSE, SIGNS AND SYMPTOMS, PATHOLOGY AND TREATMENT.

BY JAMES CANTLIE, M.B., F.R.C.S.,

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*Synonyme.*—Psilosis,—bare tongue,—from the sign most readily observed.

*Definition.*—Sprue may be defined as a catarrh of the alimentary canal, from the mouth to the anus, accompanied by marked tenderness of the tongue, diarrhoea of a special character, and associated with an atony and physiological incompetency of the organs and fluids of digestion.

*Distribution.*—The disease is met with among Europeans dwelling in China, the Straits Settlements, and in several parts of India. More careful records will no doubt reveal the fact that it is met with in many other parts of the tropical world.

*Etiology.*—The immediate cause of sprue is still a matter of dispute. Many theories have been put forward, but up to the present no one of them finds many followers. Being a tropical or sub-tropical ailment, we of course find that malaria is held to play an important part; this is a mere statement, for of malaria as a cause we have no proof. Dr. Begg, of Hankow, China, maintains that intestinal worms, more especially the round worms, are the cause of sprue. The writer thoroughly tested this belief, and cannot agree with its premises. In India there is a disease closely allied to sprue which is, or rather was, assumed to be peculiar to European dwellers in the hills. When first described it was stated that it was to be met with only at great heights, but recently more careful observations have rendered it doubtful if it is a condition affected by elevation at all. The theory is that, in the mountain streams, the particles of mica, in the unsettled and turbulent water, when drunk without filtration, set up an irritation of the mucous membrane

which resulted in a diarrhœa of a peculiar nature, termed "Hill" diarrhœa. This, like other theories, wants proof.

The writer is of opinion that the cause of sprue will be found to depend upon the food that is consumed. The disease is so essentially that of a digestive disorder that it might be presumed the cause would be in the direction of alimentation. That some irritating substance among the ingesta is the cause seems highly probable. The staple food of the European in the tropics, however, differs but little in anything but quality from that consumed by the more northerly resident, and it would seem a hopeless task to assign any one variety of food as the undoubted cause. One dish more largely used than any other, perhaps, is curry. Curries are prepared in many ways, but the native cook is generally permitted to use his own methods in its preparation. All native cooks, unless prevented doing so, will use vegetable oils in which to cook their food and, if they can manage it, that of their European employers. Now, crude vegetable oils, of native produce more especially, are acrid to a degree, and well-nigh impossible to digest. They use vegetable fat instead of animal fat because it is cheaper for their own diet, and unless their European master or mistress looks into their ways, they will sell the animal fats that are supplied to them and use their own vegetable oil instead. The smell of native cooking is an odor by itself, and is the pet aversion of all persons who have native servants. The pungent and acrid fumes given off always cause a remonstrance on the part of the European, and the cook-house has not unfrequently to be removed farther from the dwelling-house. We have evidence also from so observant an authority as Dr. Nansen, that in his Arctic trip "a pemmican made of powdered meat and cocoa-nut oil is a substance scarcely capable of digestion, and even his very dogs refused it after a time." Cocoa-nut oil itself is too expensive for the native, in most places, to use in cooking; but instead he uses a vegetable oil which is even more crude than that obtained from the cocoa-nut. The lower classes in Japan use oil made from rape-seed as a cooking medium; it is very coarse and pungent, and not used by the better classes. The Chinese use the oil expressed from the ground- (pea-) nut, and even the oil of the tea-oil-plant, a species of camellia. The pea-nut oil in small quantity is added to most dishes, and in curries more especially it forms a pretty constant quantity. Not only will the Chinese cook use this

for cooking his own food, but, unless prevented by a careful master or mistress, he will use it to cook food for the European. The tinned butter or dripping handed out to him is sold, and their places supplied by a vegetable oil as being cheaper. No doubt the daily irritation inflicted on the mucous membrane by a hot curry will have a deleterious effect on the digestion, but the addition of acrid and undigestible oils will add to the pernicious nature of the ingesta. In the opinion of the writer this is the starting-point of sprue.

*Signs and Symptoms.*—A premonitory catarrh of more or less severity ushers in all attacks of sprue. A craving for food, with a feeling of faintness, comes on, it may be, one hour before the next meal is due. The feeling speedily gives way by the ingestion of even a small quantity of food or drink, and the opposite condition of distended fulness promptly ensues. But a further development is met with in sprue to distinguish it from ordinary dyspepsia. It is found that the tongue becomes tender not only to mustard and hot curries, but anything very salt or very sweet is intolerable. Bread, even, is dreaded on account of the pain induced, and the patient is driven to consider very carefully what can be taken so as to avoid irritating the tongue. This state of things is seen to be accompanied by a raw-looking condition of the edge and part of the dorsum of the tongue. Small blisters breaking down to superficial ulcers render the side of the tongue tender to touch, ulcers of an exquisitely painful character develop in the angles between the cheeks, the lips, and the gums. As many as three or four of these are met with at once, and a succession of crops appear for some days. This untoward condition continues for about fourteen days at a time; the ulcers gradually healing and the tongue losing its tenderness. After an interval, it may be a few weeks or months, the condition is again repeated, and again disappears, with or without treatment except in a general way. There is at first no diarrhœa, the trouble seeming to confine itself to the mouth, fauces, and stomach. In the course of time, it may be a year or so, the bareness of the tongue persists and the tenderness never wholly departs. Occasional attacks of diarrhœa prevail, but only at the usual time of going to stool, or occasionally there may be two stools about the usual time, one after the other.

With these conditions present the disease may now be said to be established when the following signs and symptoms obtain: The tongue looks bare, as though stripped of its covering; the only part



where any coating may be seen is in the neighborhood of the circumvallate papillæ. The papillæ themselves stand up red and swollen in the midst of the slight coating; the fungiform papillæ are plainly to be seen swollen, pink, and flattened. The under surface of the tongue, the floor of the mouth, the gums, and the inside of the cheeks are, like the tongue, bare, red, and swollen. The tonsils seem small, the fauces red, and the back of the pharynx bare and shiny. Dyspepsia, as judged by the pain, is not so constant as one might expect from the nature of the ailment; but diarrhœa now supervenes with a characteristic stamp upon it. The patient is called out of bed early in the morning, at say five or six o'clock, by a desire to go to stool. This is followed by another stool at say seven o'clock, or at any rate before breakfast; after breakfast, and again at eleven o'clock, stools are again passed, but from thence onward, during the day, no more calls are experienced. It is purely a matinal diarrhœa, except in the very last stages of the disease that is encountered, the patient being quite comfortable after eleven o'clock in the forenoon. With the continuance of the trouble the patient loses flesh, the subcutaneous and abdominal fat is speedily absorbed. The skin becomes dry and harsh, and folds and lines appear, more especially on the neck and the region of the groins. The temperament of the sufferer alters by becoming irritable and peevish to a degree.

Withal the appetite is good, and a craving for all varieties of unwholesome food, more especially, is a prominent symptom. Sleep is not interfered with except in the early morning when the desire to go to stool calls the patient up. As time goes on and the disease remains unchecked, one finds that the patient is called out of bed to go to stool at about two o'clock in the morning. This feature once established soon becomes a habit and a prominent sign of the disease, and adds greatly to the patient's discomfort.

With sprue fully established, the patient becomes a chronic invalid and more or less of a hypochondriac. Unless it is wisely treated by change of residence and careful diet the disease may last for years, and the patient, now a little better, now worse, goes gradually down-hill and becomes incurable. The stools passed by sprue patients are of a special character. They resemble soft mud in consistence; they are light in color, well-nigh white, but mottled with a faint tinge of yellow. They are not bulky or fermented as a rule, though occasionally they are both. They are not attended by much

wind or straining or tenesmus while passing, defecation being easy and speedy.

The urine is slightly below the normal in quantity; it is light in color and of rather low specific gravity.

An examination of the abdomen will show the shrunken state to which its contents are reduced. Its walls are retracted, the spine can be readily felt through its attenuated surface, and the cavity is pronouncedly empty. The liver cannot be felt by palpation, and percussion shows its dulness to be limited to about one or two inches in the vertical line. The spleen cannot be felt, nor can, as a rule, percussion reveal its presence. The intestines are shrunken in their bulk, as their presence cannot be made out by palpation. All the abdominal viscera are attenuated and reduced to minute size. This condition is primarily, no doubt, a consequence of the disease, but more largely is it caused by the treatment,—the milk diet to which the patient is submitted. It will be seen later that it is this condition of attenuated and shrunken viscera that demands the careful attention of the practitioner who would treat his patient on rational grounds.

With these signs and symptoms fever has to be noted, and it is this feature which causes some to think that the disease is malarial in origin. But it is not a malarial variety of temperature that one finds. In the first stages of the disease "feverishness" exists, the temperature keeping at about 101° F. for weeks. The increase in body heat is followed by a depression, and for a long time the temperature may remain throughout the day a degree or two below the normal. Towards the end of the ailment, that is, before a fatal issue, the temperature rises to 103° or over, and will remain at that height for an indefinite period with but little variation.

A fatal issue is only prevented by care and treatment. If the disease is not checked the strength becomes less and less, the ability to walk about, or to exert oneself, becomes impossible, and œdema of first the feet, then the ankles and legs, becomes more marked. The heart grows feeble, and the patient dies of exhaustion.

*Pathology.*—The pathological anatomy of sprue is soon stated. The body appears emaciated to the last degree; the fat of the body, generally both internally and externally, has been absorbed. The condition is summed up in the term "death by starvation;" for such it is in appearance and in reality. The thorax seems contracted, the

heart is small, its walls atrophied, and its inner lining blanched white. The lungs are pale and the pleura glistening, with a quantity of straw-colored serous effusion in the cavity. The stomach is reduced in all its measurements, and its walls thinned; the intestines are diminished in calibre, and their coat becomes thin, white, and transparent. The liver is but half its wonted size, and the spleen is similarly diminished in its area. No organ shows any sign of active disease; they are all shrunk and shrivelled, but without any real pathological change. It is only when the intestine is opened that any departure from the healthy condition of things is to be seen. Here the mucous and submucous layers, which seem to have amalgamated, are all too easily stripped from off the general wall of the gut. The inner lining of the stomach is similarly affected, being but loosely attached to the muscular coat beneath. The bared walls of the intestine are but a fraction of their true thickness. This is the only marked abnormality to be met with in an uncomplicated case of sprue, and in reality it sums up the post-mortem appearances. The general bloodlessness of all organs is remarkable, and the portal and pulmonary systems seem as if blanched and drained of blood.

*Diagnosis.*—Sprue resembles no other disease if we except chronic diarrhœa and “Hill” diarrhœa. In chronic diarrhœa the stools are more frequent and irregular; they are fermented and more bulky; there is, moreover, in chronic diarrhœa no tender tongue, nor is the oral cavity so bare. “Hill” diarrhœa is possibly the same disease, and more careful observation of that condition will likely bring the two into line.

*Prognosis.*—Sprue, unless carefully treated, is a fatal disease. Usually, however, the patient, by care, can prolong life for many years, even if allowed to remain in a tropical climate. Patients afflicted with sprue who can afford to return to a temperate climate, whilst as yet the disease has not obtained too great a hold, may expect, with care, to wholly recover.

*Treatment.*—The general principles of treatment of sprue may be gathered from the foregoing account of the disease. Removal to a temperate climate seems to be well-nigh imperative if a permanent cure is to be brought about.

In the tropics, however, the condition can be combated and the patient restored to a fair state of health, more or less permanent in character.

In the treatment of this disease drugs play but a small part in bringing about a healthy state of the alimentary canal. Rhubarb is the only drug that seems to give any good results, but its place is quite secondary; arsenic is used empirically, but without any specific effect. What patients are to eat and drink, and how food is to be administered, are the great considerations, and everything else is secondary. The usual plan of treatment with sprue, as with all cases of intestinal flux, is to put the patient on milk. I would at once condemn this method of treatment and pronounce against it with all vehemence. The effect of milk is to bring about a stool which seems more natural, because it is more near the normal in consistence, and to reduce the number of motions. Here the good, if it is good to do so, ends; and the patient may go on with a stool which seems normal, and even increase in weight, without the disease being in any sense "cured." The stool is of light color owing to the absence of bile in the motion. Milk does not excite the liver to action, hence the functions of that organ are in abeyance, and its size, small already, grows still less under its exhibition. The stool that is passed is not a fecal stool in any sense. Analyses of its constituents will show it to be little more than a mass of soft, cheesy material. It does not smell of fæces, and in no way is this saponified mass fecal. Under this treatment the patient continues to pass one stool it may be a day, and he, or she, is enabled to get about and even to do a fair amount of work. The moment, however, any departure from a pure milk diet is attempted the diarrhoea returns, emaciation again sets in, and the patient runs down-hill quickly. Return to the pure milk diet will check the diarrhoea for a time, again to return when it is given up, or varied. It is plain, therefore, that milk diet alleviates the symptoms of sprue, but it does not cure. It frequently happens that patients may take milk for twelve months, or even much longer, to find themselves no nearer a permanent cure at the end of all the long term of self-denial.

The cure of sprue must be sought in other ways and by other means. The writer after considerable experience of this conventional method of treating sprue has given it up, and with excellent results.

The atonied and physiologically atrophied state of the chylipoietic viscera are no doubt at the root of the difficulty experienced

in the cure of sprue. The liver is reduced in size, and the persistent absence of bile in the stool shows that one of its functions is in abeyance. As with the liver so with the other viscera; they are reduced in size and function by the disease itself, and milk is not the food that is to call their activity into existence. Not only so, but the prolonged administration of milk renders the patient more difficult of cure subsequently. Milk is therefore not only no cure, but it has even a baneful effect, by its holding visceral action in a state of temporary paralysis. Any treatment which does not take cognizance of the necessity of developing the latent functions of the chylipoietic viscera must necessarily fail. Milk is not adopted as a diet for adults in health by any section of the human race. Tropical natives less even than the more northerly dwellers use milk as a diet; in fact, few of them drink or use milk in any form. The medical practitioner, however, turns at once to milk as the essence of his treatment in almost all cases of acute illness. That may be, and perhaps is, sound in theory and practice whilst disease is acute; but to attempt to live on milk, as sufferers from sprue are condemned to do for a prolonged period, is without precedent in health or in the cure of disease. Milk will not stimulate a functionless liver to action; it does not in any way call its digestive properties into play, and it but loads the liver-cells with fatty materials which they have not the power to deal with. What, then, will call upon the liver to act, and with the liver the other organs, the stomach more particularly? Starches will not, and we are therefore thrown back upon albuminoids as the only diet left. By the exhibition of nitrogenous foods alone can we hope to re-establish a healthy digestion. Beef juice made from the fresh beef, not the prepared article, will be found essential as a diet to commence with. Scraped beef, raw if need be, to be speedily supplemented by minced beef itself, raw or but slightly warmed. On this diet the writer has again and again treated sprue, and with marked success. As an adjunct to this principle of a meat diet the following rules are to be followed: The patient is to be confined to bed for a few days. A wet cold pack is to be applied to the abdomen for two hours, night and morning. If the two o'clock in the morning stool persists, the wet pack is to be applied directly after it is passed. The good effects of this will become speedily apparent. If the patient is very ill and weak the confinement to bed must be continued correspondingly, but if

the disease has not placed the patient in a very low state, a day or two will be sufficient. From the first, the patient, if only chronically, and not alarmingly, feeble, may be placed upon minced meat, to be given in quantities of five ounces at a time and three times a day. Between meals the patient is to have meat jelly to stave off hunger, and during the night, especially, is it necessary to see that some jelly is always available. Never allow the patient to be hungry. In a day or so the diet may be varied, by giving a steamed pigeon, or some winged game, similarly cooked. After a few days a baked apple may be added to the diet, once or twice a day. If all goes well, rice thoroughly boiled, and then dried by steaming, may be tried with good results. Stewed celery, sea-kail, and such vegetables are by and by to be added, until in a few days it will be found that a cut off the joint can be taken and relished. Even in the early stages the white of an egg may be given with the minced beef from the first, and soon a poached egg in its entirety will be easily digested.

This system of diet the writer has followed for years with excellent results, and maintains that it is at once practical and physiologically correct. The very first meal of a "meaty" nature will show the motion to be dark in color and bile-stained. Pent up so long, there is a slight tendency of the bile to cause looseness, which, however, must not induce the practitioner to change his treatment, as it is only the purgation caused by the return of bile to its natural channel which is causing the action. The atrophied liver by this treatment will be found to increase in size, and the practitioner can pronounce the cure to be a permanent one only when the liver resumes its normal size. In cases of great weakness food may have to be administered every fifteen minutes, and the time intervening between "feeds" increased as strength is regained.

As a drink, hot water, before and after meals, is the best. As the patient improves, rice tea, made by roasting rice brown in the oven, and then pouring boiling water over it, affords a drink at once nourishing and wholesome.

## ATONIC OR NERVOUS DYSPEPSIA AND ITS TREATMENT BY INTRAGASTRIC ELECTRIZATION.

BY A. D. ROCKWELL, M.D.,

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ATONIC is really but another term for nervous dyspepsia. Both are characterized by motor insufficiency rather than by marked disturbance of the secretory function of the stomach. The derivation of the term atony so clearly indicates its meaning that it seems hardly necessary to say that it simply implies a lack of tone or vigor of the body generally or of some special organ. I have been somewhat surprised, therefore, on more than one occasion to find that it was interpreted as an actual atrophy or degeneration of structure, —an error not alone of the laity, as evidenced by the interesting case to be related. Atony, from *a priv.* and *τόνος* tone, is a morbid condition that may affect almost any organ, but more especially those that are contractile, and of all the contractile organs atony of the stomach is the most common and the most depressing in its results. In these cases of atonic or nervous dyspepsia the most rigid investigation generally fails to show the slightest evidence of any structural change, and the symptoms can only be ascribed to enervation, sometimes general, sometimes only local, resulting in enfeebled muscular movements, and sometimes, perhaps, in altered gastric secretion. Both secretion and peristalsis are under the direct control of the nervous system. The most familiar and obvious illustrations of this are seen in the flow of tears or the sudden arrest of the saliva, the result of fright. Not only may secretory processes be arrested through nervous stress and strain, but it is easy to believe that the same cause may result in chemical changes that deteriorate the quality of the secretion. The gastric fluid thus deranged both as to quantity and quality fails to meet the necessities of the digestive process, and fermentation and putrefactive changes may occur with their train of distressing and persistent symptoms.

The form of atonic dyspepsia about which the books mainly speak, and which constitutes perhaps the most frequent example of digestive disturbance, is quite different both as to its symptoms and treatment from the atonic dyspepsia of nervous origin to which reference is here made. There is an atonic dyspepsia of the aged due alone to failing powers. Gastritis often results in enfeebled digestion, and again it may be an hereditary entailment like gout with no neurotic association. In the type of nervous dyspepsia here considered there exists digestive disturbance without any change in the chemistry of the stomach, or, at least, any that is appreciable to the ordinary tests. It is possible that, as our methods of chemical examination become more refined, we shall be able to detect errors of secretion and anatomical changes which now escape us, thus narrowing that class of gastric derangements that we now group as nervous. Just as it is beginning to be recognized that all conditions of pain and disturbed sensory states may be due to structural changes too minute for detection and capable of quick repair, so with these abnormal digestive symptoms; they may show no chemical change simply because such changes are not revealed by known methods of precision. It is interesting, therefore, in this connection to note that various careful observers have found chemical changes in cases of dyspepsia supposed to be purely nervous in character, while others have found in the same class of cases even pronounced anatomical changes.

“Jurgens has made an important contribution upon this point. In forty-one patients who, while alive, had complained of vague dyspeptic disturbances, a complete degeneration of Meissner’s and Auerbach’s plexuses was discovered; in this way he gave a tangible anatomical basis to these cases of dyspepsia, many of which had been diagnosticated as reflex dyspepsia. Furthermore, where the disturbance was more of a sensory character, he found a degeneration of the muscularis mucosæ of the stomach and of the intestines also, and a pronounced formation of varices in the intestinal walls, the exact examination of which revealed a degeneration not alone of the muscular fibres of the veins, but also of the sensory nerves and of the branches of Meissner’s plexus in the vicinity.”<sup>1</sup>

Nervous dyspepsia is almost invariably found among the well-to-do or so-called better classes, while ordinary gastric atony, the

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<sup>1</sup> Ewald, Diseases of the Stomach.



atony of another type, is found equally among the rich and the poor. It has been said that one-half the world is dying because of too much food, and the other half because of too little. Food taken in too great quantity and too frequently is the commonest cause of atonic dyspepsia among the rich. I have known cases where food has been taken every two or three hours, in the belief that it was necessary to overcome debility and inertia, the fact not being recognized that the exhaustion was in great measure due to the constant tax imposed upon the stomach and the consequent imperfect digestion and malassimilation.

On the other hand, insufficient and innutritious food is a factor quite as important in producing among the poor a very similar condition of affairs. There is a very large number who live mainly on tea, soup, and a small amount of bread. The result is that the mucous membrane becomes relaxed, with disturbed gastric secretion and loss of tone.

The diagnostic points of difference between atonic dyspepsia due to faulty diet and the atonic dyspepsia of neurotic origin are not so very marked so far as mere local symptoms are concerned, yet sufficiently so to aid somewhat.

It is well known that in the more painful affections of the stomach, such as ulcer, cancer, and inflammatory conditions, there is great pain on pressure. In nervous dyspepsia there is also invariably more or less pain on pressure, but this pain or simple uncomfortable sensation is decreased rather than increased when the pressure becomes firmer and constant. In the atonic dyspepsia of the aged or from dietetic indiscretions light pressure does not, as in nervous dyspepsia, cause any unpleasant sensation, while deep and firm pressure may and generally does occasion a distinctly uncomfortable feeling. In nervous dyspepsia it is also interesting to note the frequency with which the oxalates and phosphates appear in the urine.

Those who from overstrain from any cause find themselves sufferers from dyspepsia and nervous exhaustion will be found in the great majority of cases to furnish examples of an over-production of oxalate of lime in the economy. There is perhaps no more capricious set of symptoms than those associated with nervous dyspepsia, and in this they differ most distinctly from the symptoms, ever present and the same, that characterize the ordinary forms of

dyspepsia due to causes dietetic and local. They come and go without manifest cause. As a rule, the greatest distress is before meals, and efforts either mental or physical on an empty stomach are out of the question. Eating a hearty meal is sometimes a source of great and instant relief, while at other times it affords no relief. Hours and sometimes days intervene with a sense of well-being and an absence of gastric symptoms that greatly encourages both physician and patient, when suddenly without traceable cause all the bad feelings come back again. Cases occasionally occur in which the dyspeptic symptoms entirely disappear for a time, only to be replaced by symptoms referable to other and distant organs, and when these disappear the gastric symptoms return. I recall one patient who had for years suffered much from excessive pain and tenderness on pressure over the solar plexus three times a day before eating. At irregular intervals, however, these phenomena would temporarily disappear, but were invariably replaced by great weakness of the eyes and pain in the head. But above all else in diagnostic importance, true nervous dyspepsia is almost invariably associated with functional disorder of the general nervous system. Nervous dyspepsia is only one of the many symptoms that go to make up the neurasthenic state, and in the study of neurasthenia I have notes of two classes of cases as they relate to the disorders of digestion.

1. Where the dyspepsia precedes for a longer or shorter time the onset of general neurasthenic symptoms.

2. Where it is either coincident with the appearance of these symptoms or becomes developed long after.

It is certain that this observation has aided me not a little in the management of these cases, for in the first class my experience teaches that direct and special local treatment is far more efficacious than general methods, while in the second class, treatment directed to the general nervous system, following the ordinary therapeutic measures for neurasthenia, is most effectual in modifying the gastric symptoms. This brings me to the case to which reference has been made, and which is an illustration both of that class of cases associated with neurasthenia in which the dyspeptic symptoms precede for a longer or shorter time the neurasthenic symptoms, and of the necessity in the management thereof of direct local treatment.

A. B., a physician, came to me with the following history: His early life had been passed in hard work on a farm. At the age of

twenty-two and with perfect health he began the study of medicine, but during his course he indulged in sexual intercourse and alcohol to excess, and twice contracted gonorrhœa. After graduation he soon married, settled down to practice, and quite reformed his habits. For five years he was busily engaged in work that took him over a wide extent of territory and necessitated much irregularity in meals. He smoked incessantly during these rides, and his one other dissipation was in drinking strong tea. To these two habits he attributed his digestive troubles, although for a long time he observed absolutely no ill effects. The general nervous system seemed apparently uninfluenced. For some years after the appearance of perverted gastric sensations he was strong of nerve and buoyant in spirits.

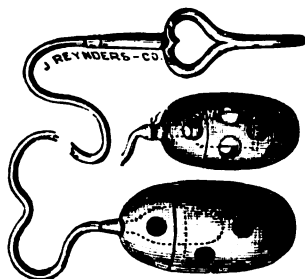
He first began to be annoyed by sensations of weight in the stomach, and by an ill-defined feeling of oppression and burning that extended into the chest and even into the throat. Finally to these symptoms were added tasteless eructations, flatulence, occasional attacks of hiccough, and every few days he quietly vomited, several hours after eating, the contents of the stomach, which seemed to have undergone absolutely no fermentation. Five years after the appearance of gastric disturbance the symptoms of neurasthenia began. These increased gradually in severity and in number, so that when he came under my observation he was suffering from morbid fears, mental instability, and in many ways was a thoroughly typical neurasthenic. He was afraid to meet people or to be alone, and had so lost confidence in himself as a physician that he was afraid to prescribe the simplest remedies. An acquaintance of his told me that he would sit for many minutes with pencil in hand, fearing to write the needed prescription lest he should prescribe the wrong drug.

In other ways also his naturally rather loose moral code was replaced by a morbid conscientiousness. He had been told, which was quite true, that he was suffering from atony of the stomach, and from that moment regarded himself an incurable. Although otherwise an intelligent man, he took it to mean that there was atrophy or wasting of the organ, and when it was explained to him that the word atony was quite different in derivation from atrophy, he expressed surprise and relief, yet could not divest himself of the idea that the condition of the stomach was beyond hope. Various examinations of the contents of the stomach showed a constant excess

of hydrochloric acid, aiding to differentiate it from the ordinary gastric disturbance associated with and dependent upon neurasthenia, and as there was no considerable secretion of mucus or other symptoms indicative of an inflammatory condition, my diagnosis was that it was simply a form of neurosis, and was the cause rather than the result of the neurasthenic state. From the fact that the gastric trouble preceded the neurasthenic by a number of years, this theory seemed not to lack rational confirmation.

An additional point of interest in connection with this case has been "the report of a physician at Kissingen, who has had occasion frequently to observe patients sent there for the waters with the diagnosis of gastric catarrh whose affection was merely a form of neurosis with excess of hydrochloric acid. These cases were fre-

FIG. 1.



Intra-gastric electrode (Einhorn).

quently the result of the abuse of tobacco, coffee, or tea, and was aggravated by the saline waters. The hydrochloric acid was found constantly in excess without much variation, in which it differs from the gastric form of neurasthenia."<sup>1</sup>

The treatment of the dyspepsia associated with and dependent upon neurasthenia is the treatment of neurasthenia itself. One of the most common results, and sometimes the first observed of the successful treatment of neurasthenia, is an improvement in the gastric symptoms, but no strictly local treatment will at all influence the general nervous condition and but slightly, if at all, the gastric. This patient had been treated for neurasthenia for a long time, but had experienced absolutely no benefit, and I determined to waste

<sup>1</sup> Quoted by the Journal of the American Medical Association, May 15, 1897, from the Berliner Klinische Wochenschrift, No. 7.

no more time in the administration of drugs or the external application of electricity, but to begin at once with intragastric electrization, and for this purpose I used the stomach electrode devised by Einhorn, illustrated in Fig. 1.

The method of procedure is simple and as follows: About two hours after a light breakfast, the stomach being quite empty, the patient would drink a glass of water and immediately swallow the electrode, which is but little if any larger than illustrated in the cut.

The advantage of this electrode, as readily seen, is that no metal touches the tissues, the current being transmitted through the holes in the hard rubber covering and through the water to the secreting surface of the stomach.

The faradic current was alone employed, and was used daily from five to ten minutes at each *séance*. From the very first application the results were beneficial. These results were not increasingly rapid, and the recovery has not been complete, but enough was accomplished in a treatment extending over two months to illustrate the decided value of the treatment and its applicability to these special cases of nervous or atonic dyspepsia that precede rather than follow neurasthenia. The gastric sensory symptoms of oppression and burning first showed signs of amelioration by no longer being felt in the chest and throat, and finally became very much less annoying in the stomach itself. The vomiting and hic-cough entirely ceased, while the tasteless eructations and flatulence were present only occasionally and in slight degree.

In regard to intragastric electrization as employed in this and other similar cases, experience has taught me that better results are obtained by the use of the high tension coil than by those in ordinary use. The current from a coil of seven thousand five hundred feet results in far greater sedation, both local and general, than the current from coils of two thousand feet or less. This fact has long been observed in the treatment of irritable uterine conditions, and is equally true as relates to the stomach. It is a mistake, also, to hold the indifferent electrode in the hand. In this way we fail to make a sufficiently strong impression upon the stomach. Long before a sufficient strength of current for this purpose has been reached the sensation in the wrist becomes unbearable. It is better, therefore, either to sit upon the indifferent electrode or apply it by means of

a broad pad to the nape of the neck (cilio-spinal centre), or back, or pit of the stomach. If in withdrawing the electrode from the stomach a resistance is felt because of an involuntary contraction of the œsophagus, as is sometimes the case, one should not pull with force, but tell the patient to swallow once or twice, when the electrode is readily withdrawn.

While the introduction of the gastric electrode is a very simple matter, no more difficult, indeed, than swallowing a large bolus, yet one often finds it no easy matter to persuade nervous patients to make the attempt, and in some cases, with the will to swallow it, there is such a nervous dread that the act becomes impossible. In these cases the difficulty can only be overcome by using a solid flexible stem instead of a string to connect with the electrode, so that it can be introduced without the aid of the patient.

## MALARIAL FEVER IN INFANTS AND CHILDREN.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY FLOYD M. CRANDALL, M.D.,

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GENTLEMEN,—In the study of pædiatrics we find comparatively few diseases which occur only in infancy and childhood. The manifestations of many diseases, however, at different ages are so varied as to make them seem like totally different conditions. This is the case with malarial fever. It was formerly thought that it was rare in infancy; it is now known that it is comparatively common. This difference of opinion is due to the peculiar manifestations of the disease during the early years of life. I shall refer this morning, therefore, only to the peculiarities of malaria in infancy, and shall take for granted a knowledge of its cause, pathology, and symptoms in the adult.

Malarial fevers are due to the development in the red corpuscles of the blood of the *plasmodium malariae* of Laveran. This parasite does not differ materially in its behavior and life history in infants and adults. The pathology is, also, practically the same at all ages. The most important pathological condition engendered by malaria is anæmia, due to wide-spread destruction of the red blood-cells and enlargement of the spleen.

The peculiar clinical manifestations of malaria are admirably shown by the two children before you.

The first patient is a girl fifteen months old, who comes from Long Island City. Her mother has tertian fever, from which she has suffered at intervals for several years. Eight days ago she noticed that the child was feverish in the afternoon. Since that time she has had fever daily, lasting for about three hours. It has not appeared at the same hour every day, but has been noticed as early as ten in the morning and as late as three in the afternoon. There

has been no chill, but preceding the fever the child has had a pinched and blue look and the hands and feet have been cold. As this condition has developed the child has become drowsy and very heavy, so much so as to alarm the mother. It has not been a natural sleep. Following the fever there has been a slight stage of sweating. The mother is clear in her statement that it is much less profuse than in her own case and that it occurs later in the day, even when the fever has occurred early. The appetite is poor; the bowels are constipated. The child in the interval, though comparatively well, is extremely irritable and hard to manage.

The child, you observe, is decidedly anæmic and has a yellowish muddy complexion. Examination shows the lungs and heart to be normal. Physical examination, in fact, reveals nothing abnormal except an enlarged spleen. By palpation, the spleen is detected at the free border of the ribs. Standing on the right side of the child and pressing the outer edge of the right hand under the ribs of the child's left side, you can readily feel the edge of the spleen. It slips away from under the hand as you press upward. Should the disease go unchecked for a week longer, you could no doubt feel the spleen below the ribs and detect the splenic notch.

The clinical history of this case would be ample to make a diagnosis of malarial fever, but we are fortunately in possession of confirmatory evidence, furnished by a blood examination. The child was brought here two days ago, and the examination of blood taken at about twelve o'clock revealed plasmodia in abundance. On that day the temperature at one o'clock was  $104.1^{\circ}$  F. Quinine in medium doses was ordered, but did not materially affect the paroxysm yesterday; the dose will be doubled to-day.

The second child is twenty-seven months old. The present symptoms began to show themselves definitely six or seven days ago. Each day the child has had a fever, coming on between ten and twelve o'clock in the morning and lasting till the middle of the afternoon. It has increased in severity from day to day. A stage of blueness was observed for the first time yesterday. There has been but little sweating. During the past three days the child has had an attack of vomiting and severe pain in the stomach with the onset of the fever. Yesterday there were three loose stools in the afternoon, but prior to that time there had been constipation. For several days the child has slept heavily. Even before the symptoms were so



definite the child would lie down and fall asleep of its own accord in the middle of the day. It has usually been difficult to get him to sleep during the day. In the latter part of the day he seems exhausted and is very listless, but in the early morning is quite bright and well.

Physical examination reveals a few bronchial râles, but nothing else abnormal in the chest. The spleen can be felt somewhat more readily than in the first case. The child, you observe, is sallow and anæmic and has an extremely listless air. A blood examination just reported reveals the plasmodium.

The first peculiarity which you undoubtedly notice in these cases is the behavior of the paroxysm. In the adult in cases of the severity shown by these children the sequence of chill, fever, and sweat is constant and characteristic. Neither of these children give the history of a chill. A chill is, in fact, extremely rare under five years. Instead of a true chill, the child becomes blue and the features are pinched; the hands and feet are cold; the skin becomes shrivelled and mottled or studded with small elevated points. This may continue from five minutes to half an hour. Even this appearance may be absent, but close observation will usually reveal at least a vestige of a cold stage. Vomiting is so common during this stage as to be almost the rule. Sharp epigastric pain is also common.

The third stage is, also, less well defined than in the adult. Sweating is rarely profuse, and is often lacking entirely. When present, it sometimes shows a peculiar tendency to delay. In the adult the sweating stage follows the fever very promptly; in the infant there is frequently a considerable interval between the two stages. In older children this stage is sometimes as well marked as in adults.

The stage of fever is, therefore, the only constant stage of the malarial paroxysm in the infant. Even this stage frequently shows peculiarities. The temperature is prone to range very high; a temperature of 104° or 105° F. or higher is not uncommon. In my experience, however, children bear these high temperatures comparatively well. The symptoms present during this stage are those common to all febrile conditions,—flushed face, suffused eyes, rapid pulse and respiration, and great thirst.

Nervous symptoms are comparatively prominent in the malarial

fevers in young children. The paroxysm is sometimes ushered in by a convulsion. This, however, must be comparatively rare in this locality. I have seen it in but two cases in a considerable experience with the disease. In one of these a convulsion occurred with the onset of the cold stage on three successive days. Physicians practising in malarial regions of the South and West have told me that convulsions ushering in a paroxysm were not uncommon in their experience. Twitchings, restlessness, severe headache, and neuralgic pains are common. Pain in the head, stupor, vomiting, and fever may lead to the diagnosis of tubercular meningitis.

Symptoms referable to sleep are extremely common. In some cases the child is very restless and wakeful during the paroxysm, but more frequently there is a peculiar heavy sleep, often amounting to stupor. The natural sleep at night, however, is usually broken and the child is restless and wakeful. In children over four years of age terrifying dreams at night are not uncommon. I have seen two well-marked cases of night-terrors which followed each daily paroxysm and ceased when the paroxysms ceased.

Enlargement of the spleen is a very important symptom of malarial infection during early life, for that organ increases and diminishes in size more readily than in adults. After the disease has continued a few days, enlargement can usually be detected if an examination is made during the paroxysm. At the end of a week it can usually be detected during the interval. In infants, percussion is of but little assistance in determining splenic enlargement. If the spleen is sufficiently enlarged to be of diagnostic importance, it can be felt at the free border of the ribs. A little experience in examining for the spleen will render detection not particularly difficult.

Anæmia is a constant accompaniment of malarial fevers of all varieties, and is due to destruction of red blood-cells by the plasmodium. Liberation of pigment at the time of segmentation produces discoloration in addition to the anæmia. Infants seem prone to acquire a sallow, yellowish hue rather than the bronzed appearance so characteristic in the adult. In severe and long-standing cases they acquire a decidedly cachectic appearance.

Bronchitis is of frequent occurrence among infants and children suffering from malaria; the appetite is fitful and is usually impaired; the child is irritable and requires unusual attention. Diarrhœa is extremely common, but constipation is more frequently seen. Hæmaturia occasionally occurs in severe cases.

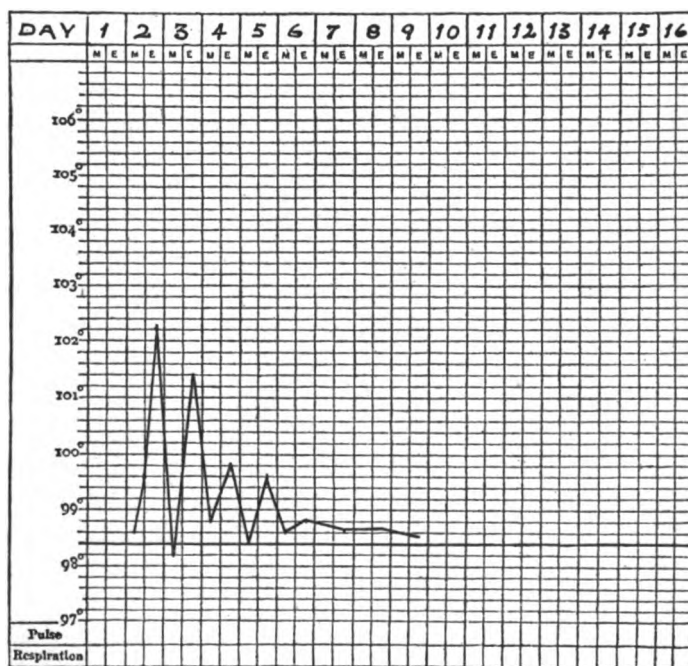
The most marked peculiarities of malarial fever under five years are as follows: 1, Mildness or absence of the first and third stages; 2, irregularity of the hour of paroxysms; 3, constant enlargement of the spleen; 4, tendency to nervous disturbances.

Acute malarial fevers usually assume the intermittent type. In very young infants and in children in whom the disease is unchecked it sometimes becomes remittent. The paroxysms usually become less clearly defined as time passes. Under four years tertian intermittent is rare, but becomes more common with increasing age. The quartan type is unknown in young children. The invariable type in infants is the quotidian,—that is, the paroxysms occurring daily. It is, in fact, however, a double tertian, for study of the plasmodium has demonstrated that the disease is due in most cases to two sets of germs, one set maturing and segmenting each day. Very irregular types of intermittent and remittent fever are often seen in infants and are very difficult of diagnosis.

Infants and children as well as adults are subject to chronic malarial infection. In this condition there is but little fever; anæmia and emaciation are marked and progressive; the skin is dry and becomes of a parchment-like color. The digestion is often deranged and the child is never well. The spleen is always enlarged, and often attains an enormous size; in older children it may be tender and painful. Dropsy sometimes occurs, and petechial eruptions are not uncommon.

Microscopical studies of recent years, as well as more accurate clinical observations, have shown the comparative frequency of malaria in young children. It has been demonstrated by several observers that the disease may even be congenital. I recently reported a case in the *Polyclinic Journal* confirmatory of this statement. For ten days the mother of the infant had suffered from tertian fever and the child was born at the end of a severe paroxysm. When eighteen hours old the child was heard to cry in a peculiar wailing manner and was found to be cold and blue. This was soon followed by fever, which was repeated on the following day, and again on the third day. The paroxysms ceased under quinine treatment. At the same time two older children had tertian fever. Examination of the blood of both mother and infant, made by Dr. James, revealed the plasmodium of malaria. The accompanying chart shows the temperature range in the case of the infant.

The diseases which are most commonly mistaken for malaria are chronic intestinal indigestion, tuberculosis, septic infection, and, in older children, typhoid fever. Demonstration of the plasmodium renders the diagnosis at once certain. This, however, is the work of an expert, and is yet unavailable for most practitioners. It is only in rare cases that the careful study of the symptoms will fail to reveal the true nature of the disease. The treatment test is a very important one, for the fact is amply demonstrated that quinine



Temperature range in a case of malarial fever in an infant eighteen hours old.

relieves acute malarial fevers, while it has but a transient effect upon non-malarial fevers. While I have called your attention to the fact that malaria is more common in young children than was formerly thought, I wish to warn you most strongly against the other extreme of loosely attributing all febrile conditions in children to malaria. In most localities there are ten cases of irregular fever due to gastro-intestinal derangements to one case due to malaria.

It is best to begin the treatment of malaria by free catharsis, calomel in small repeated doses being especially indicated. Quinine

is the only drug upon which reliance can be placed. It is well tolerated by children as regards its physiological effects, but in many cases is not tolerated by the stomach. Young children are prone to vomit it, even when it is swallowed without objection. It must be administered to them in solution. The taste is well covered by syrup of yerba santa. When not vomited this is one of the best vehicles for use. It may sometimes be administered successfully in equal parts of simple syrup and simple elixir. A solution in water will sometimes be found to be the best for young infants. Very young children may sometimes be taught to swallow a pill. The question of administration is then very simple. I hardly need to add that quinine pills should always be freshly made. If the dose required is small, tablets of quinine and chocolate may be employed. When properly made they are very palatable, but their strength is so small that they are not to be relied upon when prompt action of full doses is required. If for any reason it is impossible to administer the quinine by the mouth, it may be given by suppository or rectal injection. Irritability of the rectum in infants is the chief objection to this method of administration. In cases of necessity four grains of quinine dissolved in one ounce of water may be given by high injection through a tube. The oleate of quinine, either by inunction or by rectal injection, has been unsatisfactory in my experience. In rare cases it is necessary to administer it hypodermically.

Children require comparatively large doses of quinine, but it is difficult to give definite dosage. In deciding upon the amount to be given the daily quantity should be determined rather than the single dose. A child of one year may safely take five to eight grains daily. It may be necessary to considerably increase this amount. At three years the initial daily dose may be nine grains, to be increased as indicated by the symptoms. After ten years adult doses may be required.

Quinine is most effective if administered about three hours before the paroxysm. In the case of children it is usually best to divide the dose. I commonly give half the desired dose at seven o'clock in the morning and the remainder three hours before the paroxysm is expected, if it occurs at an hour to permit such an arrangement. In infants the paroxysm is so irregular and indefinite in its occurrence that this rule cannot be followed. It is then necessary to give a dose three times a day. The first dose may be

given at six or seven in the morning and repeated in three hours. This usually forestalls the paroxysm. A dose is again given in the evening.

In the chronic forms of malaria, quinine alone is not sufficient to effect a cure. A small amount should be given daily, but the chief reliance should be placed upon arsenic, which should be given in moderate doses for a considerable period. Reduction of the enlarged spleen is sometimes hastened by the use of iodides. The syrup of the iodide of iron is particularly indicated. Acetanilide is of decided service in relieving neuralgic pains. The anæmia in both acute and chronic cases usually requires active treatment, and it is advisable to begin the administration of iron as soon as the paroxysms have been broken.

## THE PHYSICAL EXAMINATION OF THE LIVER.

CLINICAL DEMONSTRATION, AT THE BUFFALO GENERAL HOSPITAL, TO THE SENIOR  
MEDICAL CLASS OF THE UNIVERSITY OF BUFFALO.

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GENTLEMEN,—We have met this morning for practical training in methods of physical diagnosis as applied to the liver. The patients have been taken at random from the ward, and may or may not present abnormalities of the liver itself. It is to be hoped that we shall find most of them free from evidences of hepatic trouble, since the ability to obtain practical results from a physical examination depends largely on prior familiarity with normal conditions. It must also be remembered that it is much easier to pick out an instance of marked disease than to be able to tell a patient that he has not a trouble which he or you may have suspected.

At the beginning of the lesson, I want to throw out one or two hints regarding the pathology of the liver which have a direct bearing on the results to be obtained physically. Almost all the signs and many of the symptoms of liver-disease are due either to the backing up of blood in the portal system or to a similar obstruction of bile, and, so far as the organ itself is concerned, physical examination can rarely answer any other question than "How large is the liver?" For this reason the examination of the liver lacks the interest which attaches to the heart and lungs, which have been demonstrated to you by gentlemen in other lines of practice, since the latter organs are both normally and abnormally sound-making organs, whereas the liver, with rare exceptions, is silent.

On inspection of the abdomen, what signs of hepatic trouble shall we expect? "Jaundice" is correctly answered, but if we analyze jaundice in its various forms, we must admit that it is a sign of biliary obstruction rather than of true hepatic disease. Prob-

ably the majority of *all* cases of marked yellowness are due to catarrh of the duodenum, and the majority of *serious* cases of jaundice are caused by catarrh or calculi or other obstructions to the ducts themselves. This catarrh, however, may extend up into the hepatic canaliculi, and it may be coincident with degeneration of the true liver-substance. Nevertheless, the jaundice itself represents a biliary rather than an hepatic lesion. From histologic reasons, it happens that there are two hepatic diseases that scarcely fail to produce some pressure on the biliary radicles or on the larger tubes. Do you remember which they are? Cancer is mentioned, the other is biliary hypertrophic sclerosis. There are two points in terminology to which I want to call your attention. Cirrhosis and sclerosis have a similar sound and mean practically the same thing, but as it is the hardening rather than the yellowness of the liver that constitutes the disease, it is more logical to speak of this change as sclerosis. Again, you will find many patients who will say they have never had jaundice but who will admit having had "yellow janders."

You have recently seen in the ward class a typical case of hepatic sclerosis, how would you characterize the complexion of that patient? (Answers: "Slight jaundice," "Icteric," "Sallow.") It seems to me that *sallow* is the appropriate term for the mild pigmentation of the skin and the faint tinge of the sclerotics seen in such cases, and this is the characteristic color of patients with hepatic sclerosis. Sometimes, in chronic malaria, with hepatic sclerosis, we note a decided brown pigmentation, affecting the entire body and noticeable on section even in the viscera. It is doubtful if this pigmentation is dependent on the liver changes. My attention is also called to the matter of liver-spots. These are frequently seen on the faces of middle-aged and elderly women, but also on other parts of the body, and in men. They certainly do occur when the liver is diseased, but they are also termed *maculæ uterinæ*, and they probably depend on a general dyscrasia. By the way, these spots are sometimes mistaken for the disease caused by the microsporon furfur. The latter is easily cured by parasiticide ointments.

A dry and scaly skin with a tendency to *eczéma* (don't call it *eczéma*) not infrequently accompanies sclerosis. We must recognize a sort of relationship between suppressed gout, eczema, asthma, and hepatic and renal sclerosis. There is one other condition which is quite rare, but, I believe, really more characteristic than any of these



other skin-diseases. If you should find a person whose skin is covered with scratch-marks without some obvious cause, such as insect-bites, friction of rough clothing, urticaria, eczema, etc., especially if the person is rather advanced in life, a tentative diagnosis of pruritus would be in order. Although pruritus is usually classed among nervous or skin-diseases, it is in many instances a manifestation of lithæmia, and, therefore, very likely to be connected with imperfect hepatic chemistry if not with actual sclerosis.

This patient, though not a genuine example, may serve to suggest to you another manifestation of hepatic trouble to be found by inspection. Two or three of you have at once noticed the prominent external veins of the abdomen, which suggest a damming back of portal blood and an attempt on the part of this blood to escape by way of the anastomoses between the mesenteric and external veins. Sometimes a knot of veins around the umbilicus becomes distended, and is fancifully called a *caput medusæ*. Sometimes internal veins, as those of the œsophagus, become varicose, and this condition, of which only about thirty cases had been reported up to a year ago, may terminate fatally by rupture. I have recently reported what I believed to be a case of this sort, a fatal hemorrhage having occurred at the time set for more thorough investigation of the stomach by the aid of the tube. Fortunately, the attending physician was unable to keep his appointment to call for me. Although the proof of a post-mortem examination was wanting, it seemed quite certain that the well-marked hepatic sclerosis was the cause of the hemorrhage. In this patient, as frequently happens, the portal obstruction was not seen in the superficial veins. There is another venous dilatation due to portal obstruction which may be considered either as internal or external and either as a sign or a symptom, since the patient often appears for treatment for its relief rather than because he suspects the underlying cause. This sign is, as one or two of you have guessed, hemorrhoids. This patient whom we have used to suggest the vascular signs of hepatic disease is a young man convalescing from a mild bronchitis; he has been in good general health, his complexion is normal, and his abdomen is thin and muscular. His liver, moreover, is of normal size. How are we to explain the imitation of the serious condition of circulatory obstruction? I want to emphasize this point, for this degree of circulatory disturbance has quite a typical etiology, and is of importance from the hygienic stand-point if not from that of the therapist. I scarcely expected that you

would guess the import of these distended vertical veins. Asking the patient if he has been in the habit of wearing suspenders, he says that he has usually worn a belt, whose effect may be compared to that of tight garters about the legs. The same condition may be seen in women who have suspended heavy skirts from the waist without the equalization of pressure afforded by a corset, but as they usually exercise less violently than men and are more apt to take on abdominal fat, the dilated veins are not so noticeable. In emphasizing constriction of the waist as a cause of fulness of abdominal veins, I ought to say that the last time I tried to develop this point before a class the patient denied that he had ever worn a belt, and the real cause was not discoverable. This teaches us that it is wise not to use the words always and never in regard to medical subjects.

There is only one other point in regard to inspection that I care to make. This second patient has the round, protuberant belly of intestinal indigestion, with accumulation of fæces and gas. Such conditions, especially in the middle-aged, frequently depend on hepatic sclerosis, though the sign is suggestive, like most of the rest, rather than diagnostic. Occasionally you can see the outline of an enlarged liver and, theoretically, the thrill of an organ pulsating from venous or arterial engorgement.

Palpation is of negative rather than positive value in the examination of the liver unless there is a considerable enlargement, such as cannot be demonstrated to you from these patients. In palpating the abdomen for other organs than the liver, it is best to have the patient on the back with the legs drawn up. For the liver this attitude is available, but it is often more convenient to have the patient stand and lean a little forward to relax the muscles. Use the edge of your hand rather than the fingers, but if you must use the fingers,—and some of us are obliged to on account of differences in tactile sensibility,—do not drive the nails into the flesh, or the reflex contraction of the muscles will prevent anything like a careful examination. The normal liver is palpable in the substernal triangle, and the margin of the right lobe can just be touched. In making this examination some of you have gotten the impression of an enlarged liver, because you have not discounted the thickness of the abdominal wall, which has made the margin of the liver seem just so much lower than it really is. A simple method of approximating the thickness of the abdominal wall is to pick up the tissues and divide the thickness of the double fold by two. Palpation, to be effective,

must be forcible yet gentle. Cold fingers, quick motions, even an expression of countenance that will alarm the patient, may thwart your efforts. A very nervous patient may be made to relax the abdomen while breathing quickly, or you may order him to breathe deeply and slowly, following each expiration with a firm pressure and taking advantage of the instantaneous relaxation between expiration and inspiration. The spleen is not palpable unless greatly enlarged. The gall-bladder, if much distended or hardened with stones, may be felt just below the costal arch, and external to the rectus muscles, in a line directed upward from the umbilicus at an angle of about 45 degrees from the vertical.

We will now try percussion, which is, on the whole, the most valuable method of physical examination as applied to the liver. For practical purposes we are limited to the right lobe, which normally extends from the costal arch to the level of the third rib, the summit of the dome being so far from the chest wall that it cannot be located by physical examination. I see that most of the class have marked out the upper limit of hepatic dulness at or near the sixth rib in the nipple line and the lower border at the costal arch, except in one patient, in whom intestinal tympany begins half or three-quarters of an inch above this line. This upper line is correct if you are considering the level at which the lung terminates and the liver comes into actual contact with the chest wall. But it is possible to demonstrate the normal liver by heavy percussion as high as the fourth rib. Separating my left index and middle fingers widely, so that the former lies just above the region of hepatic flatness while the latter is well up beyond any possible interference with resonance by the liver, and percussing quite forcibly, I think you can all distinguish a decided difference, although by light percussion the superficial resonance of the lung is elicited in either case. Now, as the pleximeter fingers are gradually approximated, the difference between the percussion sounds becomes less, but it is still appreciable with the two fingers lying side by side so as to overlap the fourth rib. This is normally the highest demonstrable level of the liver. In women apparently with normal livers this line often coincides with the fourth space, or even the fifth rib. Whether the difference is due to the influence of corsets or not I do not know. You will observe that, in men, the nipple affords a convenient landmark for the fourth rib, but it must be verified by actual observation in each case and even on each side of the body.

We must not be too hasty in concluding that this patient who shows intestinal tympany above the costal arch has a contracted liver, as distended intestine under the narrow edge of the liver will give tympany, just as the stomach makes it almost impossible to demonstrate by percussion the readily palpable part of the liver in the substernal triangle. By deep palpation I find that this young man's liver is as low as it normally should be, in spite of the tympany. The problem naturally presents itself, how could we make the necessary distinction in the case of a patient with rigid or thick abdominal wall? By ordinary percussion I know of only one way to obviate this difficulty,—namely, to make several examinations, perhaps administering digestives and antiseptics so as to diminish intestinal flatulence. Even then I have found tympany several times and have been on the point of making a diagnosis of hepatic sclerosis, when I happened to find the intestine sufficiently collapsed to allow the liver to be demonstrated as low as it normally should be. The distinction can almost always be made at one examination by auscultatory percussion, a method which we will pass with the mere allusion now, but which I will demonstrate to you later.

In examining the liver we must not forget the spleen, which is a tributary organ through the portal vein, and which is likely to respond in size to changes in the pressure of the portal blood. Hepatic sclerosis may go on to quite a marked degree without producing perceptible changes either in the size of the liver or the fullness of the superficial vessels. In such cases the damming back of blood is quite apt to cause an enlargement of the spleen, not so great as to become palpable, but so that the area of dulness becomes easily demonstrable by ordinary percussion, and occupies twice the normal area by auscultatory percussion. Practically the spleen is a very useful organ, simply because of the light which it throws on the liver, though I would hardly go so far as to teach this to my physiology class as a function of the spleen.

Auscultation of the liver, except with percussion, is rarely a diagnostic procedure. But we must remember the possibility of demonstrating a hydatid tumor by miniature ballottement of its contained cysts, the thrill being either felt by the fingers or heard through the stethoscope. Again, speaking without actual experience, I would call your attention to the fact that tricuspid regurgitation or possibly a direct arterial cardiac murmur may be communi-

cated to the liver or spleen so as to be audible, palpable, or visible. But I can assure you that you will listen in vain for such transmission in some of the most marked cases of valvular disease.

In the examination of the gall-bladder, the stethoscope—with the small chest-piece—is quite a valuable diagnostic instrument, as calculi will not infrequently cause a crepitus.

You will have gathered that the physical diagnosis of the liver is by no means as definite and satisfactory as might be desired, and that it is not comparable in acoustic interest with the study of the heart and lungs. In fact, often we can only say whether the liver is or is not of normal size, and we can at most judge its pathology from this fact and the amount of interference with portal and biliary currents. But for this very reason it is important that we should make the best of our limited diagnostic powers and supplement them with inductive logic drawn from ample experience. It requires comparatively little skill to make a correct diagnosis of heart and lung diseases,—with certain exceptions, in which the sounds are masked or are never distinct,—it is in the case of organs which do not express themselves plainly to our ears and eyes that diagnostic acumen is necessary.

It is well to bear in mind a convenient though by no means accurate classification of hepatic diseases, according to the size of the organ and the presence of jaundice. A very small liver is characteristic of fatty metamorphosis, an extremely rare condition, even including cases of phosphorus-poisoning. A very large and symmetrical organ suggests amyloid degeneration or perhaps leucocythæmia. A single globular enlargement is to be associated typically with hydatid cysts and abscess. A bossillated enlargement is significant of cancer. Moderate degrees of diminution correspond to atrophic sclerosis and moderate degrees of enlargement to hypertrophic sclerosis. Comparatively slight enlargement may attend congestion or catarrh of the intrahepatic biliary ducts. Ascites alone is characteristic of atrophic sclerosis, jaundice alone of cancer and catarrh, jaundice with ascites of biliary hypertrophic sclerosis. There seems to be another form of hypertrophic sclerosis which differs from the atrophic only in the excess of connective tissue. Not one of these statements can be depended on absolutely, but they may be of value as suggestions of the typical condition to be looked for in any particular disease.

## TUBERCULOSIS.

CLINICAL LECTURE DELIVERED AT THE HOSPITAL COLLEGE OF MEDICINE.

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GENTLEMEN,—This young boy, L. P., you will remember, was before the class last week, and before taking up the case in detail we will ask the gentleman who brought him here to give us a short *résumé* of the history presented by the family and by the patient personally.

He is eighteen years of age, and consulted us one week ago for the first time. He gave the history of a sore throat for one and a half years, which has been continuous for the last eight months. Father still living, and in fairly good health, but coughs considerably. Has two brothers living and in good health. Several children of the family died while infants. This patient commenced to cough about a year and a half ago; during all this time he helped to take care of his mother, who was ill with consumption; during the last nine months of her life, he was with her almost constantly. There has never been any hemorrhage from the lungs, but he has been expectorating a thick yellowish matter in considerable quantities for the last eight months.

*Physical Examination.*—On inspection we find that the breathing over the left side is somewhat restricted; there is no depression of the chest. Percussion shows dulness over the upper part of the left lung, and a cracked-pot resonance is well marked over the upper portion of the left lung. On auscultation we can get cavernous breathing and a whispering pectoriloquy. All the physical signs indicate the presence of a cavity.

*Microscopical Examination.*—We secured a specimen of the sputum and examined it carefully, and after some search tubercle

bacilli were found present in small numbers. In the microscopist's report it was not stated whether the bacilli were fully developed and vigorous, or whether they were feeble and small in size.

This is an exceedingly interesting case; it illustrates a number of features of the disease. First, this boy's inherited tendencies to the development of tuberculosis are great. His mother died of consumption, and his father has had a bad cough for a long time. We cannot say, as the mother and father are not present, what this boy's physical resemblances are, but I want to bring out the fact, in speaking of the inherited tendencies, that, as a general rule in physical resemblances, male children resemble their female parent more than they do their male parent. Apply that rule in this case and we would be warranted in inferring that this boy resembled his mother rather than his father. Of course there are exceptions to these rules, but generally we find that such is the case. We know that in a great majority of instances inherited tendencies may carry many other physical resemblances, and furthermore that the male progeny are more apt to resemble physically the mother's side rather than the father's side of the house, and with female children it is just the reverse,—they are more apt to resemble the father's side. Apply that in the case before us and we see that this boy would more than likely inherit the constitutional tendencies and tubercular trouble from his mother, who was the subject of tuberculosis. There is an argument, however, on the other side, from the fact that the mother had only been the subject of tuberculosis for a year and a half or two years. This boy is eighteen years old, and the question would naturally arise whether the mother had any of the seeds of the disease in her system at the time of his conception and birth. That is a question upon which we can throw no light. Inherited tendencies, we must concede, would be much more apt to be transmitted from the maternal parent in the active manifestations of the disease. If the parent were the subject of active progress of the disease at the time of conception, of course it is conceded that the transmitted tendencies would be greater than where at the time of conception there was no suspicion of the existence of the disease.

This case illustrates another phase of the disease,—*i.e.*, the danger of communicating the malady from one person to another. This boy tells us that he helped to nurse his mother at times, not continuously, during the first part of her illness, and almost con-

stantly during the last few months of her life, and in all probability, not knowing the importance of it, very little care was taken of the expectoration. It is likely that much of the expectoration escaped destruction. We now know, under modern scientific investigation, that it is absolutely essential to thoroughly destroy every particle of expectoration that comes from tuberculous patients, and probably in this case little care of that kind was taken, and this boy was exposed to inhalations of the dried expectoration, each particle of which contained numbers of the tubercle bacilli, for we know that this expectoration when allowed to dry upon the floor, upon articles of clothing, papers, etc., pulverizes into light dust, and floating about in the atmosphere, is drawn into the respiratory passages by every inspired breath of air. These dust particles are deposited as the current impinges against the walls of the bronchial tubes, and containing bacilli, they implant themselves and begin to work to effect an entrance into the system. During a period of eighteen months of such exposure, with a system already in a condition favorable to the development of these germs, we can hardly see how a patient would escape, and as a matter of fact during this time the germs did succeed in effecting an entrance into the system and there multiplied and developed. We have before us but the result of that development; we have infection of the system from these germs, although whether they were actually received from the mother's system or not we cannot say positively, but it seems the history of this boy shows the development of the trouble after having nursed his mother. It is possible that she may have had the seeds of the disease in a quietly progressive form in her system much longer than the history would indicate. She seems to have been confined to her bed for nine months, but she probably had a severe cough with abundant expectoration for a much longer period, and it is safe to assume that this boy received the germs of the disease from association with his mother. His system, from transmitted or inherited tendencies, was in a favorable condition for the development of any implanted germs that might subsequently be taken into the respiratory tract; then the germs, after implanting themselves upon the mucous membrane of the bronchial tubes, probably in the smaller bronchial tubes, evidently took on the process of development, gradually invading a considerable portion of the upper lobe of his left lung; then undergoing the process of degeneration, this tuber-



culous mass, at first more or less solid, has broken down and ulcerated its way into the bronchial tube, emptying the softened material in the act of expectoration, which left what we can demonstrate now, evidences of a cavity or hollow space in the upper portion of the left lung, a hollow space communicating with the bronchial tube; and this cavity, as it is called, surrounded by its pus-bearing wall, is what gives us the physical signs that we recognize to-day in a physical examination of the case.

It is important for us all to remember the group of physical signs which indicate the presence of a cavity in the lung, that the lung at some time previous has been solid, that this solidified mass has lost its vitality, has degenerated, broken down into a purulent material, that this broken-down degenerated material has ulcerated its way in the direction of least resistance, that is, towards the bronchial tubes, and emptied itself into the bronchi, and in the act of coughing has been brought up and expectorated. Now this leaves a cavity or space in the lung which gives us certain physical signs. The group of physical signs which indicate to us the presence of a cavity are: a cracked-pot resonance, elicited by percussing over the seat of the cavity when the patient's mouth is open, and when the ear of the observer is placed within easy hearing distance. When a stroke is made over the seat of the cavity and air is forced out through the bronchial tube and through the open mouth, when the waves and vibrations reach the ear they produce the sensation of a chinking noise. This is distinctive and characteristic of the presence of a cavity. There are three other sounds which we find grouped together in these cases which can be elicited on auscultation. Air passing in and out of this cavity gives us a soft, low-pitched blowing sound, which is designated cavernous breathing; the expiration is lower in pitch than inspiration, in direct contrast to bronchial breathing, which is high pitched on expiration and still higher pitched than inspiration, so that there ought to be no trouble in differentiating these. Again, we have what is called whispering pectoriloquy. If we make the patient whisper in low muffled words, as low as can be spoken, by placing your ear directly over the cavity you can distinguish the articulate words. This is known as whispering pectoriloquy. Fourth, we have what is called cavernous cough, which we can also distinguish in these cases. When you place your ear directly over the cavity and cause the patient to cough, you can

detect a distinct sepulchral sound, which occurs during expiratory coughing, and is followed by an inrush of air; in the effort of coughing the cavity is thoroughly emptied of air and is immediately refilled by forcible inspiration. As the air passes into the cavity it makes a rushing noise, which, taken in connection with the hollow, sepulchral, loud, expiratory cough, makes up what we describe as cavernous cough. It is a hollow, loud sound during expiration, followed by an inrush of air. This is also distinctive and characteristic. We have, then, four distinct points of differentiation,—viz., (1) cracked-pot resonance; (2) cavernous breathing; (3) whispering pectoriloquy; (4) cavernous cough. These four physical signs indicate to us positively, without any peradventure, that there is a cavity present in this boy's chest. The cavity is in the upper portion of the left lung, which we have located with absolute certainty.

There are several points of importance that this case ought to impress upon us. First, the absolute necessity and importance of hygienic care of a tuberculous patient in order to minimize the danger to those who are in attendance upon the case. There is not much danger so long as the expectoration is in a moist condition; the chief danger lies in allowing the expectoration to dry and become converted into pulverized dust. As long as the expectoration can be kept in a moist condition until it is completely destroyed, the danger is reduced to a minimum if not absolutely *nil*. An excellent plan is to use little paper cups designed for that purpose, made so as to be folded up in a very convenient way. The only objection to this is that it entails some expense upon patients. Of course, if patients are able to stand this expense, it is one of the most convenient things that can be used. Several of these cups may be used in a day. They are made of manila paper, do not break down when wet, they will hold water, and when filled can be thrown into the fire and destroyed in that way. If earthenware cups are used, these may be partly filled with some disinfecting fluid to prevent drying of the sputum, and after being used for a short time, the contents can be emptied into the fire, and thus completely destroyed. Destruction by fire is the most feasible and most certain method of actually destroying these germs. When that is done the expectoration is put beyond the possibility of infecting other persons. If ordinary newspapers are used as a receptacle for the sputum, these ought never to be allowed to dry. There is danger in

using anything of this kind. If the patient expectorates upon a folded newspaper or in a newspaper cone, perhaps destruction of the paper is neglected, the sputum becomes dry, little particles before we know it are wafted by the atmosphere, and other persons are exposed to danger. We have the question asked us many times, especially by couples who are sleeping together, whether there is any danger in the breath of a tuberculous patient. We might answer that question in the negative, that there is little danger in the moist breath, that all the expectoration contained in the air-passages is in a moist condition, and infection has not been actually known to occur from this source, that the main danger lies in the expectoration becoming dry, the dried particles then being inhaled. It is only after the expectoration has left the body and has become dry that it pulverizes into this dangerous dusty form and becomes a menace to other persons. While I would not advise couples sleeping together to turn their faces towards each other so that one breathes the expired air of the other, still, so far as we are able to judge, there is not much danger in this. It would be advisable, however, for them to sleep with their backs together rather than facing each other.

Care as to the surroundings of the patient is also important. The case before us is not only dangerous to those who are attending it, but it is dangerous for any one to go into quarters that have been previously occupied by a tuberculous patient. On this matter we cannot always be thoroughly posted; many persons are moving about, living in rented houses, and we cannot always tell who lived, or the condition of life of the persons who occupied such quarters previously; but this does not relieve us from the responsibility of seeking to ascertain with as much accuracy as possible whether the quarters have previously been occupied by tuberculous cases. I would hesitate very seriously in regard to living in a room that I knew had been occupied by a tuberculous patient. I certainly would not undertake to do anything of the kind without having it thoroughly renovated and disinfected; I would prefer going into a new apartment, one which I knew had never been occupied by tuberculous cases. You cannot tell what care has been exercised in regard to destruction of the expectoration, and all you know is that the apartment may have been occupied at some previous time by a tuberculous patient who may have taken no care

whatever of the expectoration; he may have expectorated on the floor, the sputum become dry, and the atmosphere of the apartment might be impregnated with tuberculous germs. In your practice in the lower walks of life, which all of us have to experience, you will probably meet with cases time and again where you will go into the room and find on your morning visit spread out on the floor several newspapers, and masses of expectoration directed towards these papers may miss them and be deposited upon the floor. You will find that the housewife will probably hurry through the task of straightening up the house; she will remove the papers that have been spread around for use during the night, upon which the patient has expectorated during the entire night. She will go through the process of sweeping, and probably spread out on the floor several masses of sputum, which during the course of the day would become dry and converted into dusty particles, and then as she passed the broom over it again would be raised in the dust and floated about in the atmosphere. This is what you will meet with time and again in your daily experience, and while such a condition of things exposes the family, you must also remember it exposes yourself to infection. I would not take any more breaths than I could possibly help in such an atmosphere as that.

The next point illustrated by this case is the further confirmatory examination that the gentleman in charge of it has made in the laboratory. While we were perfectly satisfied of the nature of the trouble from the physical examination made and the history of the case, yet we wanted a still further confirmation by microscopical examination. A specimen of the sputum was submitted to a careful investigation in the laboratory, and tubercle bacilli found. While they were not detected in large numbers, that does not preclude the fact that they may exist in large numbers. The failure to find but a few of the bacilli may simply have been a coincidental fact. While the expectoration may contain a large number of germs, the drop of sputum placed upon the slide coincidentally may contain only a few, and numerous slides may have to be examined and a careful search may be required to discover even a few tubercle bacilli. There are instruments and methods for facilitating examinations of this kind, which make it very much more easy and certain that we shall discover the germs when they are present. One is a little centrifugal instrument, very simple in mechanism, which

contains two conical-shaped tubes, into which the sputum liquefied can be placed, and when revolved at a high rate of speed the solid particles (germs), being of greater specific gravity than the liquid in which they float, will be thrown to the outer circle by the rapidly revolving tubes placed in the instrument; this serves to rapidly precipitate the solid particles, so that a pipette picking up a drop of the sediment at the bottom of the tube, if any germs are present in the entire mass of sputum they will be found at that point, and the rapidity with which the examination may be made greatly facilitates the work, and the instrument is exceedingly useful. It enables us to discover any germs that may be present, and at the same time do so in a very much more rapid way than if we simply liquefy the expectoration and then allow it to settle, which takes perhaps several hours, and with the thick liquid in which the germs are floated it would take a number of hours for them to be deposited by the natural force of gravitation. Here we can add to this force by rapidly revolving the tube, which throws the germs to the bottom and gives a precipitate in a short time.

The result of this microscopical examination confirms the diagnosis we made by physical examination, but without waiting for confirmation we placed the boy upon a plan of treatment which we have mentioned before,—that is, the creosote treatment. The use of creosote is based upon its germicidal action. It is destructive to these germs, and it is only when it is given in sufficient concentration that the germicidal influence of creosote can be felt. For that reason, in administering it we commence on small doses, and gradually increase the quantity introduced into the system until we reach the point, as it were, of saturation, until we have completely saturated all the tissues and all the fluids of the body with creosote, which then is brought in contact with the germs in sufficient concentration to prove destructive to their vitality, and in this way good is accomplished by the administration of a remedy like this.

There are other methods of treatment, but the administration of creosote in hot milk I believe to be the best plan that I have ever pursued. For many years I have given it in ordinary capsules, but the objection to that is that you cannot increase the dose beyond a certain limit, which sometimes falls short of effecting good. In the ordinary No. 3 capsules you can drop about twelve to fifteen minims of beechwood creosote. Be careful always to secure a pure form of the

drug; the ordinary commercial creosote is too irritating to be efficient. Beechwood creosote is the best form, and it can be given in capsules after eating; you can increase the dose up to twelve or fifteen minims without any unpleasant symptoms, and in most cases when you reach that limit you will note the beneficial effects from its use. In some cases, however, you will find that you cannot give this quantity without its giving rise to some unpleasant sensations, due to the creosote coming in contact with the membranes of the stomach in a too concentrated form. We obviate that to some extent by giving it after meals. Always have the stomach filled with a meal, then when the capsule dissolves and the creosote is liberated it is taken up with the rest of the food, and of course only comes in contact with the mucous membrane of the stomach in a dilute form. But the better plan, I think, and one which enables us to increase the dose greatly beyond the usual amount that is taken, is the administration of creosote in hot milk. Take a teacupful of hot milk, drop the creosote in and stir it; the effect is to break the drug up into very small globules; it becomes emulsified with the milk. These small globules are mixed with the milk just as butter is mixed with milk before it is churned, and it makes a smooth emulsion, and when taken into the stomach in this form we do not get the burning or pungent effect. In this way we can increase the amount gradually, drop by drop, until I have had some patients take as much as fifty or sixty minims of creosote three times a day. When you reach a point like that the whole system is permeated with the creosote, fluids as well as solid tissue, and we find the emanations from the body all tinged, giving off the odor of creosote, so we cannot go into a room where the patient has been taking creosote in this way without perceiving the suggestive odor of this drug. When given to that point we may expect some beneficial effect upon the germs themselves, and when a patient is taking it in this way the expectoration changes in character, and the whole feeling of the patient is altered and changed. There is less fever, less expectoration, and an improvement is soon manifested.

I wanted to speak to-day of other plans of treatment, for instance, the serum treatment, or what is called the Paquin treatment. The anti-tubercular serum treatment is exceedingly interesting, and is based upon the principle that the serum will acquire an immunizing property, that it will acquire a property that makes it

either destructive to the toxic principle generated by these germs, or serves to stimulate and invigorate the resisting power of the system against these germs and the toxic principles that are given off by them.

When disease germs are introduced into the system, at once there is a contest for supremacy set up between the invading germs and the cells of the system. These germs not only multiply rapidly and produce local destruction, but at the same time they give off poisonous or toxic principles which pervade the system. The toxic principle that is given off by these germs in the system of course is injurious, and when this contest for supremacy is set up, then there is a constant battle for this supremacy, and if the system has great resisting power it may succeed in neutralizing this toxic principle and in finally destroying or driving out the germs from the system. If we could watch a tubercle colony that effects entrance to the system, we would find that the system fights point by point by means of a wall of cells which seek to confine the germs to that particular point, by throwing around them a wall of lymph, and then to accomplish destruction and disintegration of these germs and prevent their further invasion of the system. This is not always accomplished by the unaided efforts of nature. We find after a time the germs multiply in large numbers and gradually push back the resisting wall, make a break through it, implant their colonies in other parts of the system, or the germs break through and are carried into the surrounding vessels and lymphatics, and then through that medium are conveyed even to distant parts of the system. Then a rally is made by the resisting cells, and we have another line of defence formed a little larger to correspond in extent to the attacking army. If the system can be stimulated to renewed action by the administration of such remedies as creosote, we can lessen the vitality of the germs and render assistance to the system in this way. This stimulation may be brought about by mechanical and medicinal agents.

It has been found that the serum of the blood acquires a certain power of resistance against these toxic principles which are given off by the germs, and we find that the introduction into the system of serum that has acquired an increased resistive influence against the toxic principles generated by these germs renders just that much assistance to the system. It has been found that the serum of certain animals, the goat, for instance, as well as the dog and horse,

can, by being subjected to certain processes, be made to acquire this strong influence, and then when introduced into the human system will prove destructive to the further extension of the germs. Of course the system of the animal is first brought under the influence of the toxic principle which is acquired from cultures of these germs, the toxine is introduced first in an attenuated form, and the animal's system is able to overcome it. Then it is introduced in a little stronger doses, and still the animal is able to overcome it; then a little stronger, and so on until the full influence of the toxic principle exhibited from the culture germs is introduced into the animal's system without producing any injurious effect. When that point has been reached, then the serum is withdrawn from the circulation of the animal and is utilized by its introduction in small quantities into the human system, producing in that way a beneficial effect. There are two theories as to how this effect is produced. One is the direct chemical theory, based upon the supposition that the antitoxine that is introduced into the system neutralizes the toxic principle which has been given off from the germs. Another theory, which is perhaps more plausible, is that the introduction of this antitoxic principle derived from the animal's system merely acts indirectly by stimulating and invigorating the resistive power of the cells in distant parts of the system that are producing this antitoxic principle that is present in the body, and in this way the introduction of the antitoxine from the animal acts by simply helping or invigorating the natural resistive efforts on the part of the system into which it is introduced.

As to the results of this treatment. It is too early to know the ultimate effect or the benefit that may be derived from the use of this serum, but I have been very well pleased with the experience I have had with it, and the records that have been put in print are exceedingly favorable as a rule. Of course there are some exceptions. The earlier a remedy like this is used of course the better, which is also true of any remedy we might make use of, the earlier we take the case and bring it under treatment the better. If we take a case in the early stages of this disease, build up the system with reconstructives, and administer the remedies outlined, we may frequently accomplish a cure. And if Nature can, as she no doubt often does, accomplish a cure in some of the early cases, with a remedy like the serum mentioned, we may naturally expect much



better results. Even in the later and more advanced cases much benefit will undoubtedly be derived. We cannot expect, where the lung tissue is largely involved and broken down, forming large cavities, to see the same marked benefits, the same absolute results from the use of a remedy like the serum, as we would in the earlier manifestations of the disease.

We will continue this boy on the creosote treatment and note the progress of the case from time to time. One of the chief objections to the serum treatment is its expense; it is difficult to stand the expense of a remedy of this kind in a charity dispensary. In more wealthy patients we can use such a remedy, and I hope after a time we may be able to thoroughly familiarize you with the methods of its use and make some further comments as to the results which can be secured by its use.

# Neurology.

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## TOXIC POLYNEURITIS.<sup>1</sup>

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF PRAGUE.

BY PROFESSOR R. VON JAKSCH,

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GENTLEMEN,—We begin our work this morning after the long holidays without preface. The case for our consideration is likely to prove so interesting that we proceed at once to the superficial examination of the patient. As he lies before you you see that he is a very young man, well developed, not emaciated, with a somewhat cyanotic face and evidently rather labored breathing. What is especially noticeable, however, is the constrained position which he occupies in bed. You will notice as we proceed with the lecture that he does not alter this position, though it would be awkward and tiresome for a healthy person to retain it.

He lies partly on his left side with his head well turned to the left, and with the appearance of having a tonic contraction of the muscles of the back of the neck. You have heard one of your colleagues say in answer to my question that he looks not unlike a patient suffering from meningitis. There is, however, no question of meningitis in the case, and his position is due entirely to the fact (as is so often noted in pleurisy and pneumonia) that while lying so his breathing is easier. This must not lead you away at once to the thought, however, that we have to do with primary lung trouble, for we would scarcely begin our examination with this in view before we would be struck by a number of symptoms that point to an entirely different affection.

His face is cyanotic, but besides it is stolid and expressionless.

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<sup>1</sup> Reported by Dr. James J. Walsh, Berlin, Germany.

There is slight ptosis of the left eye, showing that the innervation of the oculomotor is disturbed. When we ask him his name he answers intelligently and intelligibly at once, though evidently with an effort, and tells us, moreover, that he is a merchant's clerk. His sensorium is unaffected, then, though you may have noticed when he spoke that there was some difference in his facial innervation on the two sides. This is brought out more clearly as I ask him to show his teeth, and it is evident that there is left-sided paresis in the facial region.

Evidently we have to do with a nervous case, and so we proceed to find out from his history how the affection began and what are his antecedents.

He is twenty years old, a clerk. His parents are living and healthy. He has five brothers and sisters; all in good health. Two others died in childhood. Up to two years ago the patient himself has always enjoyed good health. At that time he was for fourteen days in bed with red, swollen, and tender knees and wrists. The tumor, rubor, calor, and dolor, of an acute inflammation, which was evidently rheumatism.

He got completely over this, and has had no symptoms since that would indicate that other serous membranes, his endocardium or pericardium, for example, had been involved in the rheumatic process.

He continued in good health until the 26th of December, 1897, about two weeks ago, when he felt creepy, burning feelings in the fingers of his right hand, and thinks that he noted that sensation in them was diminished. Parasthesiæ are often the result of toxic influences, and there are many different toxines that may give rise to them. Ergot is especially to be noted in this regard, and ergotismus used to be popularly called the "creepy sickness," from the peculiar feelings experienced by patients in their extremities when the condition began to assert itself. Alcohol induces similar symptoms at times, but in such cases usually as the forerunner of a distinct alcoholic polyneuritis. In general polyneuritis is preceded by these paræsthesias, but so is tetany and tabes, so that much weight cannot be put on the symptom as indicative of any special condition.

The next day after he had experienced these peculiar feelings, when he attempted to get up he had excruciating pains in his back and feet, so that he could not stand up, and was compelled to lie

down again. He made a second earnest effort to get up and walk despite the pain, but his legs gave away under him and he fell to the floor. His legs trembled a great deal and he noticed that his hands and arms, especially the left one, were weaker than before when he attempted to help himself. His right wrist remained weak, and on admission there was some "wrist-drop" on this side.

This, together with some other neuritic symptoms, would make us think of lead-poisoning. Here in Bohemia of late years, especially in patients from the northern parts, from around Richenberg, we have had a number of very severe cases of lead-poisoning. Lead wheels are used in polishing garnets and other Bohemian precious stones. During the process minute particles of lead are thrown off from the wheels and, floating around the rooms, are breathed in or settle on food or drink, and so find an entrance into the body. It has become a house industry now, and at times whole families are affected. The cases are of the extremest type. It is a question whether the government ought not to interfere, and allow the industry to be carried on only with such regulations and careful state inspection and control as will protect the workmen from the serious danger of lead-poisoning involved.

Here, however, we have no history of working in lead, and he has none of the symptoms that accompany chronic lead intoxication. His arteries are as soft and yielding as is to be expected at his age. His urine contains no albumen and no casts, and is normal in other particulars. He has no contraction of his kidneys, and no hypertrophy of his heart.

Wrist-drop in the right wrist was the only outspoken paralytic symptom, though there was paresis of all his extremities when he was admitted, but a whole series of other phenomena of paralytic character have developed since. He has been before you now for some time, and yet he has not moved from the constrained position we noted when he was first brought in. The reason is not far to seek; he is practically unable to move. All four of his extremities have become completely paralyzed since his admission. The paralysis developed asymmetrically and irregularly. First his right arm became completely helpless, then his left leg, then his left arm, then the right leg. Now, when I ask him to raise his hands, he turns his trunk, showing that he has the best of good will in the matter, but does not succeed in moving his arms at all. The same thing occurs

for his legs; his hips he can sway to and fro, but an actual movement of his legs he cannot bring about. When with paralysis of all four extremities we take into account his ptosis and his evident facial disturbance, you see what a wide-spread symptom-complex we have.

The manner of development of the symptoms until the present condition was reached is extremely interesting. When he came in his history was taken, according to the custom of the clinic, very exactly, special attention being paid to the nervous system because of the nerve symptoms present. The patient is a young, healthy-looking man, of medium size and development. He lies on his back by preference. His skin is normally moist and of equable temperature. There are no atrophic lesions, no scars, and no abnormal reflexes. His head is brachycephalic. Hair dark blond and thick. His mucous membranes are somewhat cyanotic. His tonsils slightly hypertrophied. The mucous membrane of his pharynx is slightly redder than normal. His tongue is moist, with a thin white coating. His neck is of medium thickness and length, and shows the presence of no tumors and of no abnormal pulsation. His thorax is of medium size and proportionately well developed. His breathing is costo-abdominal, not frequent or intense, and is rhythmical. His apex-beat is visible under the lower border of the fourth rib and two finger-breadths inside the nipple-line. His heart action is normal; his pulse corresponds to it.

The difference between the normal regular rhythmical breathing described on admission and that we can see now is very striking. The patient is still lying on his left side, but, as you see, he is scarcely using that side to breathe with at all. *It lags distinctly* behind the right side in its movement, and its range of undulation is very distinctly less, in fact is scarcely more than just visible. With the wide-spread nervous symptoms in the case we can only conclude that there is also a question here of nervous disturbance. His left phrenic nerve is evidently included in the process which has affected so many other nerves. He lies on that side to be able to use the other side more easily for breathing purposes, and that side also appears to be affected in its activity, so that there would seem to be some slight paresis of his right phrenic, too.

As it was evidently a nervous case, the condition of his nerves on admission was carefully investigated. Each of the nerves, as is the custom of the clinic, was carefully tested.

1. Thiophene and vanillin promptly and readily recognized,—normal.

2. No narrowing of his visual fields and his acuity of vision not diminished. His color fields are normal.

3. Movements of the bulbus oculi not inhibited, pupils react normally.

4. Normal.

5. Motor branch normal, chewing movements regular, painless, effective. No pain on pressure. On the anterior two-thirds of the tongue the taste is good.

6. Normal.

7. He shows his teeth, wrinkles his forehead, and shuts his eyes promptly, fully, and without bother.

8. Hearing seems normal, and there are no subjective sensations.

9. Taste on the posterior one-third of the tongue normal.

10 and 11. There are no bulbar symptoms and no pulse disturbance.

12. Movements of the tongue normal. It is promptly projected in the middle line. There is no atrophy, no trembling, and no fibrillary contractions to be noticed. Salivation is not disturbed and he can expectorate normally. His faculty for speech and writing is not disturbed.

Both his arms are affected, though he is still able to use them, the right somewhat better than the left. The dynamometer shows that while the right hand has some slight power still remaining the left has scarcely any. There is complete palsy of both legs, and not the slightest resistance to passive motion is offered. The dynamometer here shows complete absence of power. There are no contractures, no cramps, and no tremors. There are no trophic disturbances present. The erector spinæ group of muscles is involved in the general paresis and he is unable to sit up. The muscles of the front of the trunk do not seem affected. It is impossible, owing to his general paretic condition, to test his incoördination.

He has no disturbance of sensibility nor of his temperature sense, no anomalies of his sense of touch or of pain. His patellar reflex and ankle-clonus are both absent. His abdominal muscle reflex is, however, present, and so is his cremaster reflex. His hands and feet are moist and sweaty, but not more than normal. He has no abnormal sweating, no flashes of heat and cold, no patches of cu-

taneous hyperæmia and anæmia or of œdema; in general his sympathetic system seems unaffected.

This was his condition on entrance. You see how different it is from what he describes as his condition when first attacked; yet only three days, from the 27th to the 30th of December, intervene. At first some paræsthesia, then weakness of one arm, then of both legs, with pains in his calves. In three days almost complete helplessness. The affection is eminently a progressive one, and our examination of him now will show you that this steady progression has continued.

He still lies in the constrained position we have noted before. There seems to be some left-sided strabismus, as if he were looking, according to the rule, towards the tumor in his brain. But ready-made rules for diagnosis are very apt to be misleading, especially in nervous diseases, and here this would lead us far astray. We have evidently to do with wide-spread peripheral disturbance, not with a localized central lesion. We note the expressionless condition of the left side of his face as we have him slightly turned. When we ask him to close his eyes, we note that the left fails to be covered by the upper lid; the condition being known as lagophthalmos or hare-eye, because the hairs instead of projecting out from the eyelid to meet the cilia of the lower one are inverted with the lid, and leave the white of the eye exposed. When we have him wrinkle his forehead, the wrinkles fail on the left; and when we have him laugh, the nasal and oral folds are much deeper on the right, and the mouth is drawn in that direction. We have to do, therefore, with an affection which involves all three branches of the facial nerve. This points to a peripheral cause as most probable, though not absolutely certain.

So far he has had no diplopia. When we ask him to follow our finger with his eyes, a series of phenomena develop that have not been noticed in the case up to this time, and which we must note carefully. His abducens on the right is evidently interfered with. He cannot bring this eye beyond the median line. But neither can he bring his left eye beyond the median line, though the movement outward is comparatively free. I say comparatively because all the movements of the *bulbus oculi* are limited. We are evidently assisting at the development of the symptom complex, whose significance it is the merit of the immortal Graefe to have pointed out,—paralysis of the external muscles of the eye,—*ophthalmoplegia externa*.

The development of it in this irregular fashion, with the abducens on the right and the internal rectus on the left, first completely taken, is very unusual, and we shall have our ophthalmologist make a careful examination of the eye-grounds and other ocular phenomena. His pupils react to light and to accommodation, and the ophthalmoscopic report of five days ago gave no "choked disk,"—i.e., no oedematous or inflammatory condition of the optic nerve-ending, no beginning or partial atrophy of the nerve, and no other abnormalities on the eye-ground. In cases like this especially, careful ophthalmoscopy is often of the greatest aid to the neurologist, and I cannot insist too much on attention being paid to it. Hyperæmia or hemorrhage of the retina pointing to a meningitis, or the white shining plaques, that once seen are never to be forgotten, of a tubercular choroiditis, often settle a doubtful diagnosis. Here we must get along without any such aid.

As we proceed with the examination of muscles supplied by nerves at the base of the brain we find other suggestions of a peripheral affection. The tongue is unchanged and there are no fibrillary tremors,—his hypoglossus is unaffected. He complains, however, of an increase of saliva on the right so that his chorda tympani is involved,—another sign of the peripheral nature of the affection. In the muscles of his face there is a clear reaction of degeneration. He swallows well, so that his vagus and accessorius remain unaffected, though, owing to the involvement of his phrenics, he cannot cough or sneeze.

Our clinical picture includes, then, an affection of all the nerves of four extremities,—of those of the neck and back, of the phrenics, and certain of the nerves at the base of the brain. We evidently have to do with an intoxication, but just what the noxa is that is at work we are not in a position to say. Most of the peripheral nerves have been affected by it. If one of the infectious fevers had preceded the attack by from ten days to three weeks, we would have no hesitation in attributing the symptoms to the specific toxine of the disease, and would consider the matter settled. We have, however, no such history; nothing except the rheumatism two years before; and while the tendency to contract rheumatism again after a first attack shows that some important modification of the organism has taken place that may last for years, still we would hesitate to think of the toxine of the disease stored away in the system for so long and only manifesting its presence now.



The clinical picture is not unlike that which used to be described as Landry's paralysis. Ascending paralysis it is also called, though the affection may be a descending one quite as well, and so resemble more especially our own case. It is very probable that most, if not all, of these cases of Landry's paralysis were really a multiple peripheral neuritis. The anatomical lesions described as occurring in the disease are of too varied and sometimes too contradictory a character to constitute the basis for an independent disease. And in some cases no lesions at all were found, especially no lesions of the central nervous system, above all of the cord, though it is here that the disease was supposed to exert its ravages.

One of the differential diagnostic points between multiple neuritis and Landry's paralysis is said to be the failure to find disturbances of sensibility in the latter. In our case sensation remains practically unchanged. You see that he is able to distinguish correctly and readily between heat and cold, and his sense of touch and of pain is not diminished. His abdominal and cremaster reflexes remain as at the beginning, unchanged. His patellar reflex is absent, but his muscle and periosteal reflexes in the arm are intact. His rectal and bladder reflexes are undisturbed. According to the old theory, disturbance in the cord would have influenced these by cutting off the inhibitory faculty of higher centres. We can say no more now than that there exists no disturbance of either the sensory or motor neurons to these parts, and so the reflex arc through which sensation influences motor discharges remains intact.

With the irregularly developing paralysis of his extremities alone to guide us, we might have thought of another affection in which of late we have been especially interested.

A young woman of twenty-nine came to the clinic some time ago with a distinctly septic temperature and with a series of peculiar paralytic symptoms. Various muscles and muscle groups in the arms and legs were affected in very irregular fashion. After studying the case for some time we concluded that the only diagnosis that would explain the peculiar symptom complex the case presented—the disconnected palsies, without any symmetry or localization of relation—was multiple abscesses of the cord. The autopsy yesterday confirmed our diagnosis. There is, I think, only one other case in the literature, and that is from Professor Nothnagel described some years ago. (This case will be published in detail by my assistant

shortly.) A short section of the cord in which the punctiform abscesses may be seen I shall pass around now.

Once before we made the diagnosis of multiple cord abscesses in a case presenting many similar features, but it was not substantiated by the autopsy. The patient was a man of forty, who came in with fever and with odoriferous sputum, besides a series of irregular disconnected muscular paralyses. Knowing the tendency of certain septic processes in the lungs, as the purulent bronchitis of dilated bronchi, to produce localized lesions in the central nervous system, especially the brain, I made the diagnosis of multiple abscesses of the cord and perhaps brain, but it was not confirmed by the autopsy. Had they been found in the case the origin of the abscesses would have been clear, while in our case of yesterday no reason could be found for their occurrence, no point of infection from which the micro-organisms might have been carried by the blood-stream to the cord.

As a case of multiple neuritis the prognosis is not as bad as it would be if there were multiple abscesses in the cord. Where the neuritis involves nerves that are connected with vital functions as here, then the prognosis is always serious. It is just these cases which have given the supposed Landry's ascending paralysis such a hard name and the character of a fatal disease. I have seen, however, a man in whom both phrenic nerves were more affected than they are here recover, so that the possibility of recovery is not absolutely out of the question. The disease has, however, in this case been steadily progressive, and there are no signs that it has reached its acme and is about to decline; yet it needs must be so soon if we are to have any reasonable hope of life being preserved.

Where recovery ensues convalescence is extremely slow and tedious. Such a case as this would take months, perhaps more than a year, to get entirely well. Then, too, the mere fact of recovery does not ensure complete restoration of function; and a patient may remain for years after an attack of multiple neuritis without the use of or with weakened members. With lead and alcoholic neuritis this is not an infrequent occurrence, this failure of nerves and muscles to return to the normal, and all of you must have had your attention called to such cases.

As to the treatment for multiple neuritis, it depends on the etiology of the affection. Here we have been unable to determine the

etiology, but have been led to the conclusion that the toxins causing the disease were of bacterial origin. There is the possibility of a rheumatic element in the case, so we have given him plentiful doses of salicylic preparations. Besides its antirheumatic action, if we are to assume that it has one, this drug would act as a mild antiseptic in the system generally. If the symptoms were due to degenerate forms of the streptococcus or staphylococcus whose toxins had not been sufficiently virulent to affect the system generally or produce local lesions, but still had had enough virulence to affect delicate nervous structures, sodium salicylate would be as rational and as promising a drug as we could employ.

If there were a history of syphilis in the case I would give him a course of mercurial inunctions, and should await the result very hopefully. You must remember from last year a case of very generalized multiple neuritis where the bulbar symptoms were becoming more and more threatening. He was a man of thirty-six, who denied ever having had syphilis, but who promptly improved and went on to complete recovery under inunctions of mercury.

A number of cases of so-called acute tabes are really syphilitic polyneuritis. This is to be borne in mind all the more sedulously, as the prognosis of multiple neuritis is much, ever so much, better than that of tabes. The therapy of tabes, too, is absolutely hopeless, while that of syphilitic polyneuritis is one of the most satisfactory chapters in the whole tale of therapeutics.

The important treatment, however, in most cases of polyneuritis is that of the complications induced by the primary affection. In our case here this must be our main care. Perfectly healthy lungs were noted upon his entrance into the hospital; there was a clear percussion note all over the chest and fremitus and normal vesicular breathing. We now have all over the chest râles of all sizes. The involvement of the phrenic nerves has led to stagnation of secretions within the bronchi, and this has led to diffuse bronchitis. Further retention will give rise to capillary bronchitis and to bronchopneumonia or to gangrene. Your colleague suggests in answer to my question expectorants for the condition, but expectorants are only of use when there is some expectoration and we wish to increase the amount of secretion by irritation so as to more thoroughly clean out the lung by the amount of secretion that will be coughed up. But here, owing to the involvement of the diaphragm, we have no cough

and no expectoration. Here we must try other means. We do not wish to let the secretions stagnate in the bronchi and cause hypostatic pneumonia, and so we provide for frequent changes of position. At intervals of one-half hour for to-day his position will be changed. If he is no better this evening he will be given a warm bath. Meantime he will be given expectorants too. *Hydrastis canadensis* perhaps, as your colleague suggests. Our experience in the clinic with it justifies every confidence in it, yet I prefer here the old reliable ammonium chloride. I fear, however, from the rapid advance of the lung symptoms, that remedies will be of little avail, and that it is but a question of a day or two before we shall have the opportunity to confirm at the autopsy our diagnosis of *polyneuritis toxica e causa ignota*.

[The very unfavorable prognosis given during the clinic was realized by the death of the patient on the evening of the third day after the lecture. The autopsy, made by Professor Chiari at the Pathological Institute of the German University, fully confirmed the diagnosis. No changes, macroscopically at least, in the central nervous system could be found to account for the symptoms. The peripheral nerves, however, in frozen stained sections, showed the existence of an acute neuritis. No origin for the toxine that caused the wide-spread peripheral neuritis was found. It is one of those cases of fatal toxic polyneuritis, occurring after exposure to cold, sometimes without even this indefinite etiology, which have been reported of late years, and are a tempting subject for further investigation.]

## ARSENICAL NEURITIS.

CLINICAL LECTURE DELIVERED AT THE CHARING CROSS HOSPITAL.

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GENTLEMEN,—I intend to-day to give a lecture upon a very interesting case of arsenical neuritis, but before demonstrating the principal clinical features of this case I will make a few general remarks upon this poison. Arsenic has almost from time immemorial been the poison most frequently elected for criminal purposes on account of the small quantity required to produce deadly effects, the readiness with which it can be obtained, its comparative tastelessness, and when skilfully administered to avoid suspicion, the liability on the part of the medical man to associate the symptoms with those of disease,—*e.g.*, it would be possible to avoid overlooking arsenical poisoning during a cholera epidemic, and this actually took place in Magdeburg when a number of people were poisoned, and only after the insurance company's suspicions had been aroused by the payment of a number of premiums to one man was the murderer found out. Again, the poisoner, Mrs. Flanagan, was only found out by the suspicion of the companies being aroused to the payment of premiums upon a number of infants who had died. Infantile diarrhoea from bad feeding was doubtless certified as the cause of death in these children. Fortunately for the social community, no poison is so readily detected chemically. Poisoning by arsenic may be acute, subacute, or chronic. The usual form in which it enters the system is arsenous acid (white arsenic). In about half an hour (often less) after taking a large dose of arsenic there is a burning pain at the pit of the stomach, accompanied by a sense of constriction in the throat and a metallic taste in the mouth. Violent purging and vomiting ensue; the discharges, at first mucous, become bilious and tinged with blood as in English cholera; there is excessive

thirst, the urine is suppressed, the arterial pressure falls so that the pulse at the wrist can hardly be felt, and the extremities become cold, all sure signs of the fatal collapse that will ensue. Towards the end cyanosis, cramps, convulsions, and coma supervene, and the patient dies in from five to twenty hours.

The subacute form of poisoning exhibits symptoms of a milder character, and generally they are remittent. The vomiting and purging may intermit and the abdominal pain may subside, although it is still present on pressure. The heart's action is weak and irregular, the skin cold and clammy, the urine diminished and often albuminous, while cramps and convulsions in the legs may occur, and, as a rule, the patient is conscious up to the last.

Chronic arsenical poisoning may occur occasionally from continuous use of the drug. I remember a little girl who was in the Children's Ward some years ago. She was admitted for chorea, and she had been treated for some time outside. She had only been in the hospital a few days and had not been seen by the physician; the resident house physician had given her five-minim doses of liquor arsenicalis, which treatment was quite orthodox, and the case was shown to me by him as an interesting case of paraplegia in chorea. The child had running at the eyes, sickness, a temperature of  $101^{\circ}$  to  $102.5^{\circ}$  F., with sore throat and enlarged tonsils, but no signs of a membrane; naturally these symptoms were attributed to tonsillitis. I found, however, that she had some tenderness over the epigastrium, pigmentation of the fingers and hands in the form of small oval or round discrete brown areas varying in size from one to four millimetres; complete absence of the knee-jerk, great weakness in the legs, and diminished excitability of the muscles to the faradic current, with also some diminution of cutaneous sensibility. She had no affection of speech nor of visual accommodation, therefore we could not attribute the paralysis in the legs to a previous attack of diphtheria. It was known that she had been taking liquor arsenicalis for some time; seeing that she began to improve as soon as the drug was stopped and eventually made a complete recovery, we can only attribute these symptoms to the action of the arsenic. I have heard of other cases of chorea in which liquor arsenicalis had been administered for a long time producing serious toxic effects; one case was discovered by a distinguished candidate while under examination, possibly to the annoyance of the examiner, who had

overlooked this cause of the symptoms. In prescribing arsenic for chorea for any length of time it is always well to inquire if there is any sneezing, running at the eyes, feeling of sickness, or pain at the epigastrium. The case, however, which I am now about to show you is exceptional and interesting, because the mode in which the poison has entered the system is unusual, the results are serious because the man is permanently incapacitated and the employers have not shown the necessary care in protecting him from the poison, and are therefore responsible under the Employers' Liability Act. I will narrate to you the history and clinical phenomena exhibited by this case, and then make a few general remarks upon arsenical neuritis.

September, 1896, W. M., aged fifty-six, married, three children, all alive and well. Never had illness before and of temperate habits. Occupation, cleaner of clothes at a chemical works. He commenced this work four months ago, and about six weeks after he had been at the work he noticed a numbness and tingling in the fingers of the right hand with gradual and progressive loss of power in the hand, so that he was obliged to give up his work because he could not hold the scrubbing-brush. Upon inquiry he told me that the clothes he had to clean were of India-rubber and used to protect the men who were employed in the manufacture of *sheep dip*, and this compound contained large quantities of arsenic and alkali. He used a pail of water and a scrubbing-brush, and he continually dipped the brush into the water to clean it; his right hand and arm were thus continually plunged into an alkaline arsenical solution of increasing strength as he proceeded with his work. The right hand was, therefore, much more exposed to the poison, and also had the heaviest work to do.

There was no tenderness over the ulnar nerves on pressure and the nails appeared normal, nor was there any pigmentation of the skin nor wasting of the muscles. The hand felt stiff and numb as if it had been long exposed to the cold, he could not hold a pen, and when asked to write his name he was unable to get further than this, although he had been a good penman previously. Observe the ataxic character of his writing, not unlike that of a general paralytic. He could only hold the pen when he looked at it, and if his attention was taken off it dropped from his hand.

He had considerable loss of sensation to pressure, heat, and cold;

when a small clip was fixed on his hand, his eyes being shut, he was unaware of its presence. There was a diminution of sensibility over the right forearm and hand to all forms of sensory stimulus, but especially noticeable in the fingers, thumb, and palm of the hand, and he was unable to perform delicate movements of the fingers. There was no apparent trophic change in the skin, and he said his general health was good, that he had had no sickness, pain at the pit of the stomach, nor itching and running of the eyes, nor sneezing and running of the nose. On electrical examination it was found that the reactions to the faradic current in the muscles of both arms and hands were diminished. A slightly stronger current was required to cause contraction of the muscles of the right side. There was no reaction of degeneration with the galvanic current. The next noticeable feature was the strong current the patient could bear over the right forearm and hand, showing greatly diminished sensibility. A fortnight later he came again to see me. He had been attending the electrical department; there was no wasting of the muscles, but there was still sensory paralysis. The hand felt warmer and there was a slight improvement in tactile sensibility and localization, for he could now recognize rough tactile sensations. He said that he had curious sensations in his feet.

He came again six months later and complained of trouble in passing water, having to wait some little time before a flow came. There was still the same loss of power in the right hand, the fingers and thumb were fixed in a state of semiflexion and there was very little power of movement; he could not flex the fingers and thumb to clinch his fist, and therefore he had no grasp. The forearm was semiflexed and adducted, and midway between pronation and supination; he could not raise it to a right angle. He could not place his right hand upon his left shoulder; in fact, he could not get nearer with the tips of his fingers than seven inches. It was possible that some of this impairment of movement in the arm was owing to disuse. There was paresis in both legs and a very tottering spastic gait. The knee-jerks were very exaggerated, but there was no ankle-clonus nor any marked muscular wasting in the legs. There was no jaw-jerk nor difficulty in speech or swallowing, and the pupils and fundi oculorum were normal. He felt his helpless position very much and burst into tears. *The right hand was now smooth and glossy*, a condition which had come on since last I saw him.



He has been attending, as you know, my out-patient department for the past year, and his condition is as you now see him, only altered for the worse. The sensation in his hand has improved; but he has little or no movement. The left hand is very feeble and there is general muscular weakness, the knee-jerks are greatly exaggerated, and there is ankle-clonus now obtainable on both sides. It is certain that this man has been permanently incapacitated. He will never recover the use of his right hand. He has, moreover, a progressive enfeeblement of power in his left hand and his legs; the tottering, feeble gait with exaggerated knee-jerks and slight ankle-clonus on both sides indicates a *degeneration of the cortical pyramidal neurons* progressive in character, but whether it will be followed by a progressive degeneration of the remainder of the motor path—viz., the *neurons* of the anterior cornua—I cannot say. I dare say you have remarked also that he is emotional, occasionally bursting into tears when spoken to. I think these evident signs of cerebral degeneration may be merely secondary and not in any way directly due to the arsenic; but the right arm and hand are obviously the seat of a neuritis, the glossy condition of the skin (*vide* photograph) is proof positive of this; moreover, the occupation of the patient distinctly shows that he was continually exposing the right hand and arm to the influence of the poison. He tells us that he was employed all day washing the India-rubber clothes of men employed in the sheep-dipping manufacture. The sheep-dipping consists of soft soap, arsenic, etc. He used his right hand to scrub off the adherent stuff from the India-rubber clothes, and every now and again he would dip the scrubbing-brush, his hand and arm into the bucket of water; it would not be long before the water contained a large amount of arsenic in solution; moreover, the alkali would render the skin more susceptible to its absorption; no wonder, then, that after a comparatively short time at this occupation numbness, weakness, and stiffness in the hand and arm occurred. The poison in all probability first acted upon the peripheral endings of the nerves in the skin and muscles, producing a true peripheral neuritis. He has exhibited none of the general symptoms of arsenical poisoning, nor could we detect any arsenic in the urine. It is said that arsenic is eliminated by the kidneys. I could, moreover, not detect any pigmentation of the skin, and altogether I should think it probable that the main effect of the poison has been



**Illustration of glossy smoothness of hands in arsenical neuritis**



upon the peripheral nerves of the right hand and arm. No doubt had he continued longer at this work he would have displayed the general symptoms of arsenical poisoning. It is astounding that the managers of the works did not provide him with *India-rubber gloves* to wash the *India-rubber* clothes of the workmen; it shows to my mind a strange lack of intelligence.

Perhaps some of you remember a patient that used to attend my out-patient department, E. F., employed in the telegraphic department of the General Post-Office. His work had been for years to clean the zincs of the batteries and to cover them with mercury amalgam. He had to give up the occupation because he lost the use of his hands. You will remember he had "*main en griffe*" owing to paralysis of the interossei; he had numbness in the fingers and a hypertrophied condition of the skin. This condition I attributed to his occupation, and he was found other employment by the authorities. Whether the local neuritis was due to mercury, zinc, or arsenic, a frequent impurity in the latter, I cannot say, but I should incline to the opinion that it was the constant absorption of mercury.

The treatment of this case of arsenical neuritis has been most unsatisfactory. He has had galvanism, massage, and strychnine internally,—the best we can do for him. He certainly has recovered sensibility to pressure, but the power of movement has not returned, the muscles respond to faradism requiring a stronger current than normal, and I believe the loss of power is as much due to the destruction of peripheral sensory endings as to the affection of the motor nerves. I am sure he is not malingering; moreover, he could not *malinger* away the natural ridges, folds, and lines of the skin producing that glossy smoothness (*vide* photograph). His employers have wisely decided to allow him half-pay all the while he is incapacitated. I think I shall ask one of the surgeons to see him with a view to breaking down adhesions in the joints, as possibly this might give the muscles a better chance of receiving power with massage. Cases of arsenical neuritis are generally of unfavorable prognosis. This, however, is a very unusual one.

Since I gave the above lecture another probable case of chronic arsenical poisoning has come under notice in my asylum practice. A woman, aged forty-eight, who had passed the climacteric period, had been a patient in one of the London county asylums for some

considerable time, suffering with delusional insanity. She was anæmic, and the medical officer had put her on moderate doses (five minims) of Fowler's solution; after she had been on this treatment some time she developed great weakness in the legs, with foot-drop, absence of knee-jerks, and quantitative as well as qualitative electrical changes in the muscles; there was also tenderness on pressure of the muscles; in fact, the case resembled in all particulars one of alcoholic neuritis; but alcohol as the toxic influence could be absolutely excluded, for it is certain she had had none for at least eighteen months. The conclusion was, that these moderate doses of arsenic might have occasioned this condition in a person just at a period of life (climacteric) when the nervous system is particularly liable to be affected by poisonous influences. I have noticed that women at this period of life are particularly susceptible to the toxic influence of alcohol. It is probable that arsenic acts as a tonic to the nervous system by promoting metabolic activity in the neurons, and it may be that when morbid changes occur it is due to the excessive metabolism; thus physiological balance between repair and waste (which probably varies to some extent in every individual) is overturned, degeneration of the neurons being the result.

I remember also a case of pernicious anæmia in the hospital, who was treated with large doses of liquor arsenicalis with the greatest success; all the while he was kept upon this drug the excessive hæmolysis with which he suffered was to a great extent stayed; it had, however, to be discontinued, for he showed symptoms of arsenical poisoning,—viz., vomiting, pain at the pit of the stomach, sneezing, and running of the nose, conjunctivitis, and pigmentation of the skin, and, if I remember rightly, there was a silvery scaly condition of the tongue. After discontinuance of the drug the corpuscles sank from one million five hundred thousand rapidly to six hundred thousand, and the patient died. It behooves us, therefore, not to be afraid of using this *most valuable* therapeutic agent; but to take every precaution to prevent toxic effects by watching and carefully examining the patient for the first signs of gastric disturbance or neuritis.

# Surgery.

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## THE ETIOLOGY AND CLASSIFICATION OF CYSTITIS.

PAPER READ BEFORE THE AMERICAN SURGICAL ASSOCIATION.

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GENTLEMEN,—The bladder is the dilated portion of the urinary tract interposed between the ureters and the urethra, and in a normal condition serves as a temporary reservoir for the urine. It is composed, like all receptacles of this kind, of three anatomically and physiologically distinct coats,—(1) mucous coat, (2) muscular coat, and (3) peritoneal coat. The mucous coat differs from the structures of this kind as found in most other organs in that it contains no glandular appendages. The mucous membrane of the urinary bladder is devoid of glands, and is made up of several layers of squamous epithelium. The fibres of the muscular coat are arranged in such a manner that when they contract, the organ being in a normal physiological condition, complete evacuation of its contents takes place, while during the intervals between urination they permit insensible painless distention to the physiological limits. The serous coat, like all peritoneal investments of other abdominal organs, is admirably adapted to insure free mobility of the organ in the performance of its complicated functions. The bladder is freely supplied with blood-vessels, which take such an important part in all inflammatory processes. The lymphatic vessels are found only in the muscular coat, and play an important rôle in the pathology of interstitial cystitis and in the extension of infective processes to and from the bladder.

### ABSORPTION FROM THE BLADDER.

In the study of inflammatory affections of the bladder it is important to obtain a clear conception of the function of its mucous

lining. The epithelial lining of the bladder is not, properly speaking, a mucous membrane, as it is not supplied with glands and in a normal state secretes no mucus. It is the reservoir for an excretion and not for a secretion. For this, if for no other reason, we should *a priori* question its ability to absorb medicinal and toxic substances. The mucosa of the bladder contains no lymphatics; it lacks, therefore, all the physiological elements necessary for absorption. Gerota (*Anatomische Anzeiger*, Band xii., p. 347), in the examination of more than sixty bladders, could not demonstrate, either macroscopically or microscopically, the presence of lymphatic vessels belonging to the mucous membrane. None could be detected in the trigonum. The few vessels found in the submucosa of the vesical neck were identified as the lymphatics of the urethra, which extend for a short distance into the neck of the bladder, but soon enter into the muscular coat. That the normal mucous membrane of the bladder is not an absorbing surface has been demonstrated by the clinical observations of Civiale and many other surgeons and the experimental work of many investigators, among them Kuss, Susiné, Alapy, Alling, Lewin and Goldschmidt, Caze-neuve, and Livon.

Guyon is of the opinion that the vesical mucous membrane lacks the function to absorb, while Bazy and Sabatier are of the opposite belief.

Hottinger's ("Zur Frage der Absorptionsfähigkeit der gesunden Harnblase," *Centralblatt für die Krankheiten der Sexual-Organ*e, Band vii., Heft 5) experiments seem to prove that enormous quantities of poison must be introduced into the bladder of animals to produce death. Death in such cases he attributes to a process of diffusion rather than of absorption.

Lewin and Goldschmidt ("Die Resorption Körperfremder Stoffe aus der Harnblase," *Archiv für Experimentelle Pathologie*, December 30, 1895) made many experiments on animals, and came to the conclusion that the healthy mucous membrane of the bladder is impermeable to toxic substances, and when absorption does take place it is from the prostatic portion. Their experiments were made by ligating the neck of the bladder and injecting the solution directly into the bladder through an abdominal incision.

Alapy ("Ueber das Absorptionsvermögen der Harnblase," *Centralblatt für die Krankheiten der Harn- und Sexual-Organ*e, 1895,

Band vi., Heft 4 and 5) experimented with strychnine and obtained very variable results. With a view of explaining these differences in the results, he made a further series of experiments with cyanide of potassium. All of the animals thus treated died. He concluded from his experiments that when a non-volatile poison is used and the results are not constant, the positive results followed the absorption of the poison from the prostatic portion, while no absorption occurred from the vesical mucous membrane, and that death resulted in those male animals in which the poison escaped from the bladder through the urethra.

Recently Ashdown has succeeded in killing rabbits by injecting poisonous substances into the bladder.

Bazy and Mazon, from repetition of the same experiments, came to the conclusion that poisonous substances in solution are absorbed by the intact vesical mucous membrane. It is more than possible that in these experiments absorption took place not from the mucous membrane of the bladder, but by the escape of fluid into and absorption from the urethra. From a practical stand-point it is safe to assume that the intact mucous membrane of the bladder is a non-absorbing surface, and that when absorption does take place it is in consequence of injuries and lesions which open up avenues for the entrance of toxic and infective substances into the lymphatics or blood-vessels of the middle coat.

A rational discussion of the diagnosis, prognosis, and treatment of cystitis must be based almost entirely on what we know concerning the etiology and classification of this very variable and obstinate disease. I have deemed it expedient and advisable to limit my remarks on this occasion to the etiology and classification of cystitis, with the expectation that it may serve the purpose of preparing the way for a more general and thorough discussion of the more purely surgical aspects of this disease at our next or some subsequent meeting.

## I. ETIOLOGY.

The recent great advancements in the prevention and more successful treatment of infective surgical diseases are the direct outcome of the vast increase of our knowledge concerning their etiology. Surgical bacteriology paved the way for rational surgery. It is now very generally conceded that inflammation of any tissue



or organ is invariably caused by microbic invasion, and that all other causes only act by determining or favoring infection. If this be true, it is apparent that the successful treatment of cystitis presupposes an accurate knowledge of the nature of the microbic origin of the inflammation. Suppurative cystitis and tubercular cystitis are so entirely different in the nature of their bacteriologic origin that the method of treatment successful in one would almost with certainty aggravate the other. Inflammation of the bladder is often the result of a mixed infection, and it is of paramount importance in all such cases to gain accurate information by bacteriologic examination of the urine concerning the part which each kind of microbes plays in the causation and continuance of the inflammatory lesion before an intelligent and successful course of treatment can be devised and carried into effect. It is of special importance in the successful management of cystitis and conditions mimicking cystitis clinically to make a sharp distinction between the cases in which the symptoms are caused by inflammation and those in which they are due to non-inflammatory pathological conditions. It will be seen from these remarks that the modern etiologic study of cystitis is based largely on a carefully conducted bacteriologic examination of the urine. The exercise of patience and perseverance is often required, as in many cases the urine has to be examined repeatedly before the necessary information is gained.

(a) *Predisposing Causes*.—In our old text-books we find in the discussion of the etiology of disease the familiar distinction made between predisposing and exciting causes. In our modern literature we find all the exciting causes discussed under the head of predisposing causes, and the list of exciting causes is an entirely new one, containing terms unknown to medical and surgical writers of less than fifty years ago. As predisposing causes of cystitis we recognize and describe such injuries, agencies, and influences as are concerned in establishing a *locus minoris resistentiæ* in the tissues of the bladder in which a sufficient number of pathogenic microbes of adequate virulence accumulate to produce those tissue changes which characterize inflammation. The injured tissues or contents of the bladder furnish the necessary nutrient medium in which the microbes grow and multiply. All of the predisposing causes do one of two things or both: 1. They effect tissue changes which determine the localization of microbes from the bladder, adjacent organs,

or the general circulation. 2. They furnish a nutrient medium for the growth and multiplication of microbes.

*Retention of Urine.*—The most frequent of all predisposing causes of cystitis is retention of urine from any cause. Retention of urine acts in two ways in predisposing the bladder to infection: 1. The retained urine serves as a culture medium for some of the microbes which are known to produce cystitis. 2. The distended bladder wall is subjected to pressure which in itself is a potent predisposing influence.

Mesnil de Rochemont ("Zur Pathogenese der Blasenentzündung," Leipzig, 1896) made experiments with a view of demonstrating that the presence of pathogenic bacteria in the bladder is not sufficient to produce cystitis, but that a second factor is necessary to induce inflammation which he has found in a disposition in the bladder wall. He made a bacteriological examination of the urine in twenty-five cases of cystitis. The urine was removed from the bladder under strict aseptic precautions. From these cases he cultivated from the urine fourteen different kinds of microbes. Of these microbes there were nine species facultative anaërobic and five obligate aërobic. One-half of them decomposed the urea; the remainder had no such effect. With the exception of one, all proved pyogenic in the animals experimented upon. He found that sterile foreign bodies inserted into the bladder and causing no obstruction never produced inflammation of the mucous membrane. The experiments with pure cultures were made by introducing the microbes into the bladder from above. His experiments also proved that the presence in the bladder of obligate anaërobic or facultative anaërobic microbes did not produce cystitis whether they did or did not decompose the urine. If, however, the urethra was obstructed at the neck of the bladder for twenty-four hours and the urine accumulated during this time, the presence of facultative anaërobic microbes, regardless of their effect on urea, caused cystitis, the urine remaining acid, while the presence of obligate aërobic microbes, on the other hand, which produced at the same time urea decomposition inserted into the bladder under the same conditions, caused ammoniacal suppurative cystitis. A temporary obstruction of the urethra without infection of the bladder according to duration produced hyperæmia, hemorrhages, and even necrosis.

The local effect of the toxic and irritating substances formed by

the action of some of the microbes on the urea of the retained or residual urine constitutes an additional element in the causation and propagation of inflammatory processes in the bladder. The gravest cases of cystitis occur in patients suffering from acute retention of urine in the event of the bladder becoming infected by careless catheterization, or otherwise. It is in such instances, more especially if the bladder is at the same time paralyzed, that the inflammation attacks almost from the very beginning the whole surface of the mucous membrane, and is so prone to extend along the ureters to the pelves of the kidneys. In cases of gradually increasing obstruction to the free outflow of urine, so frequently seen in neglected cases of stricture of the urethra and in prostatic patients, the residual urine plays an important rôle in the origin and extension of inflammatory affections of the bladder. It is in cases of cystitis with such a mode of origin that the trigonum of the bladder is almost constantly the starting-point of the infection and the inflammation following it. It is time that the medical profession should realize the well-known clinical fact that retention of urine is one of the most frequent and potent conditions in increasing the receptivity of the bladder to infection, and that cystitis can be most effectively guarded against by preventing the accumulation of urine in the bladder beyond the physiological limits. Knowing the great susceptibility of the bladder to infection when the urine is retained, it becomes the surgeon's duty to employ the strictest aseptic precautions in the evacuation of the organ by catheterization, puncture, or incision.

*Unrest of the Bladder.*—Abnormally increased muscular action of the bladder, as occurs in cases of central or peripheral irritation of the nerves which preside over the muscular structure of this organ, or in consequence of the action of local irritants, as stone, foreign bodies, tumors, chemical or toxic substances, is a recognized predisposing cause of inflammation of the bladder. The same may be said of reflex influences from adjacent organs, which exert a similar influence in increasing the function of the muscular fibres beyond the physiological requirements. Exaggerated muscular contractions of the bladder favor infection by the vascular changes which they produce, and especially by inducing the surgeon so often to resort to unnecessary harmful instrumental examination.

*Abnormal Urine.*—Abnormal conditions affecting the quality

or quantity of urine frequently precede inflammation of the bladder, and must be regarded in the light of predisposing causes. Hyperacidity or, more frequently, alkalinity of the urine, by causing congestion of the vesical mucous membrane and by disturbing the function of the bladder, furnish the conditions for a subsequent infection. Albuminuria, polyuria, and especially diabetes appear to increase the susceptibility of the bladder to infection. The elimination through the urine of irritants such as cantharides, turpentine, and allied drugs, and the irritating principles of certain articles of food and certain alcoholic beverages, produces alterations in the mucous membrane of the bladder which prepare the tissues for infection and inflammation.

*Tumors.*—Tumors of the bladder, malignant and benign, frequently precede and complicate cystitis. Vesical tumors may prepare the bladder in different ways for infection. They may cause retention of urine, vesical tenesmus, hemorrhage into the bladder, and, in the event of ulceration, the urine comes in contact with an abnormal surface, all conditions which would be favorable to infection. Again, tumors of the bladder are productive of symptoms which lead the surgeon frequently to explore its interior by the use of instruments, a method of examination to which the date of infection can be so often traced.

*Calculus and Foreign Bodies.*—The presence of a stone in the bladder is attended by symptoms which point to inflammation of the neck of the bladder minus the evidences revealed by a microscopic examination of the urine. It has been shown experimentally that the presence of foreign bodies in the bladder never produces inflammation independently of pathogenic microbes. A calculus or a foreign body becomes a predisposing cause of cystitis by the production of local lesions and vascular changes favorable to the localization and growth of bacteria, which are the essential cause of the inflammation. It is well known that a stone may remain in the bladder for years without causing cystitis, and the cystitis which so often complicates such cases can, in the majority of instances, be traced to instrumental examination.

*Pressure.*—Compression of the bladder from within and from without is a potent predisposing cause of cystitis. Pressure from either direction diminishes the vascularity and nutrition of the bladder wall, and in this way increases the susceptibility of the

tissues to invasion by disease-producing micro-organisms. This predisposing cause is most frequently met with in connection with cystitis in pregnant and puerperal women and in cases of myofibroma of the uterus, in which the tumor makes pressure against the bladder. It is in such instances that the infection is likely to extend from the surface to the underlying connective tissue, and that the subsequent inflammation often terminates in extensive exfoliation. An enlarged prostate may so encroach upon the bladder as to cause sufficient pressure to become a predisposing cause aside from the secondary lesions resulting from the chronic obstruction.

*Exposure to Cold.*—Sudden chilling of the surface of the body results in congestion of internal organs, which occasionally affects the bladder sufficiently to effect vascular changes, and they in turn determine an infection which otherwise would not have occurred. The embarrassed circulation in the vessels of the bladder is then to be considered as the direct cause of the localization of pathogenic microbes in this organ sufficient in quantity and virulence to produce an inflammation. In the absence of microbes in the bladder, adjacent organs, or the general circulation, no amount of exposure to cold can cause a cystitis.

*Venous Stasis.*—We have already mentioned as local predisposing causes of cystitis conditions which produce congestion, and have regarded them as predisposing causes. Venous stasis in the vessels of the bladder from more distant causes acts in a similar manner and is followed by the same results. Among such remote causes which disturb the circulation in the vessels of the mucous membrane of the bladder must be mentioned general enfeeblement of the circulation from different debilitating causes, organic heart-disease, consolidation or compression of the lungs, and cirrhosis of the liver.

*Trauma.*—To trauma has been assigned for centuries a direct influence in the causation of cystitis. Since it has been made clear that no inflammation is possible without infection, trauma has been transferred to the list of predisposing causes in the etiology of inflammation of the bladder as well as of any other organ. Trauma of the bladder from injury, the presence of foreign substances, or the action of chemical, toxic, or thermal influences creates a *locus minoris resistentiæ* for the localization of pathogenic microbes, or opens a pathway for their entrance from the bladder into the tissues

composing the bladder wall. Considered in this light, trauma in its various forms constitutes an important element in the causation of cystitis. The injection or instillation into the bladder of caustic solutions strong enough to damage the epithelial lining of the bladder has often provoked a cystitis which without such therapeutic intervention would never have occurred. The trauma inflicted by the passage of instruments into the bladder in search of stone or foreign bodies has undoubtedly been often responsible for similar disastrous results. Operations upon the bladder by either the suprapubic or perineal route have occasionally been followed by septic cystitis from infection during or after the operation, when the interior of the bladder before the operative intervention was in an aseptic condition. Contusion or laceration of the bladder resulting from the application of force from without or within never gives rise to cystitis unless the accident is followed by infection, either by the introduction of microbes by catheterization or the localization of microbes from the blood in the parts injured.

(b) *Exciting Causes.*—The essential or exciting cause of cystitis is invariably the presence and pathogenic action of microbes in the tissues of the bladder, the seat of inflammation. In the study of the etiology of cystitis it is important to consider in detail the routes of infection. From a practical stand-point it is well to admit that pathogenic microbes reach the interior of the bladder most frequently through the urethra by the use of instruments, or spontaneously by the extension of infective processes from the mucous membrane of the urethra to that of the bladder by continuity of surface. It is, however, well to remember that microbes may and do reach the bladder by other routes,—namely, through the urine from the kidney, the general circulation, and adjacent infected organs through the lymphatic channels. The study of the etiologic relationship of bacteria to cystitis dates back to a little more than ten years, and during this time sufficient proof has been accumulated to show that inflammation of the bladder is always the result of bacterial invasion. It was soon found that the disease may be produced by any microbe which possesses pyogenic properties.

Bumm ("Die Aetiologie des puerperalen Blasenkatarrhs," First Congress of the German Gynecological Society, Munich, 1886) first made use of Koch's plate cultures in making bacteriological examination of eight cases of cystitis occurring in puerperal

women. He found a diplococcus in all of the specimens examined, associated in some of them with the gonococcus or the staphylococcus pyogenes aureus. The microbes cultivated from the urine were also found in the lochia, which led him to conclude that the cystitis was caused by the use of the catheter. Cultures made from the urine injected into the bladders of dogs caused cystitis only in case the mucous membrane had been previously injured by retention of urine or the action of chemical irritants.

In 1887 Clado described a microbe which he found in the urine of patients suffering from cystitis, which he called "bactérie septique de la vessie." This microbe grew upon gelatin, which it liquefied, and rendered the nutrient medium alkaline. Pure cultures injected into the tissues of mice and rabbits produced septicæmia. Injections of the same into the bladder obstructed by ligature of the penis caused severe cystitis.

In 1889 Doyen described fourteen kinds of microbes in the urine of cystitis patients, ten kinds of bacilli, and four kinds of micrococci. Pure cultures of any of the bacilli injected into the peritoneal cavity of guinea-pigs was followed by death in from five to twenty-four hours. There is but little doubt that most of the bacilli found by French investigators in the urine of patients suffering from cystitis are identical with the colon bacillus.

In 1890 Lundström described two kinds of cystitis microbes, staphylococcus ureæ liquefaciens and staphylococcus ureæ candidus, both of which rendered the urine alkaline, besides causing the cystitis. Pure cultures injected into the bladders of rabbits, combined with temporary retention, caused cystitis of a suppurative type.

Albarran and Halle ("Note sur une bactérie pyogène et rôle dans l'infection urinaire," *Bulletin de l'Académie de Médecine*, August 21, 1888) were the first to make a systematic bacteriological investigation of the urine in cystitis. Their work is based on a study of fifty cases of suppurative cystitis, pyelonephritis, and kidney abscess. In forty-seven of these cases they found numerous bacilli; in fifteen of these, bacilli could be cultivated separately in pure cultures; in the rest cocci were also present. The bacillus was called "bactérie pyogène," and Albarran came to the conclusion, as the result of his investigations, that this organism produced the greatest number of cases of cystitis.

Melchior ("Om Cystitis og Urininfektion kliniske eksperimen-

telle og bakteriologiske Studier," 1893) found the colon bacillus (*urobacillus communis*) in twenty-five out of thirty cases of cystitis, seventeen times as an isolated microbe. The bacterium next in frequency was the streptococcus pyogenes, which was found five times, three times in pure culture. In one case he cultivated from the urine the bacillus of typhoid fever, in a patient suffering from cystitis, two weeks after convalescence. From a scientific as well as from a practical stand-point it is time to admit that cystitis is invariably a microbic disease, and that the proper prophylactic precautions consist in preventing the entrance of microbes into the bladder, more especially in cases of urine retention and when the bladder is the seat of a lesion which offers a predisposition to infection. It is necessary to consider carefully and in detail the different avenues through which the bladder is reached by pathogenic microbes which are known to produce inflammation of this organ.

*Infection through the Urethra.*—Every surgeon is familiar with the frequency with which the passage of instruments into the bladder is followed by cystitis, particularly when the urethra is the seat of inflammation and in case catheterization is performed for retention of urine and the strictest aseptic precautions are not observed. By continuity of surface a suppurative inflammation of the urethra may extend to the bladder without instrumental intervention. Complete sterilization of catheter and hands does not always succeed in depriving catheterization of the danger of bladder infection. Pathogenic microbes are almost constantly found in the normal urethra of healthy persons.

Lustgarten and Mannaberg ("Ueber die Mikroorganismen der normalen männlichen Urethra," *Vierteljahrsschrift für Dermatologie und Syphilis*, 1887, p. 405) made a bacteriological examination of the urethras of eight healthy men, and found ten different kinds of microbes, among them many which are known to produce cystitis. These observations have been fully corroborated by the researches of Rovsing, Steinschneider and Galewsky, Legrain, Melchior and Petit, and Wassermann. The meatus is a favorite lodging-place for microbes. If the meatus is not disinfected before the insertion of the catheter, microbes may be carried with the instrument into the bladder sufficient in number and virulence to provoke a cystitis, provided they are brought in contact with a soil prepared for their reception and growth by an injury



or antecedent lesion. During a recent visit to the obstetrical wards of Professor von Winckel I was informed that for some time quite a large number of the recently delivered women had suffered from cystitis. The strictest antiseptic precautions were practised in sterilizing instruments and hands, but the prevalence of this puerperal complication continued until the professor introduced an additional precautionary measure, in all cases requiring the use of the catheter, —namely, disinfection of the meatus with a solution of the mercuric bichloride. From that time on cystitis from this cause disappeared. Secondary gonorrhœal cystitis following a specific urethritis, although a rare complication, does occur, but is more prone to follow a mixed infection of the urethra.

*Infection by the Urine.*—Authors continue to insist that practically the urine is sterile, and that infection of the bladder from this source seldom if ever occurs. This statement does not agree with accurate clinical observations or the results of examination of normal urine secreted by healthy kidneys. In some cases the passage of microbes through the kidneys produces temporary lesions which appear before the urine contains bacteria.

Schweiger ("Ueber das Durchgehen von Bacillen durch die Nieren," *Virchow's Archiv*, Band c., Heft 2) has shown conclusively by his careful clinical observations that the urine from scarlatinal patients is contagious; for varicella, typhus recurrens, and malaria the same holds true. In typhus, Gaffky has found bacilli in the renal vessels. As in most infective diseases the kidneys show textural changes; it is natural to conclude that the renal lesions were caused by microbes on their way out of the body. Schweiger looks upon all kidney lesions found in the course of infective diseases as of bacillary origin. To prove that microbes pass through the kidneys, he cultivated a bacillus which Reimann had discovered in the pus of ozæna. This bacillus is stained an intense green color in a culture of gelatin and agar after twenty-four hours. The cultures of this green bacillus were suspended in a sterilized physiological solution of salt, and injected directly into the circulation. The experiments were made on a dog, a cat, and a rabbit. The bacillus did not pass directly through the kidneys, but a certain length of time intervened between the injection and the appearance of the bacillus in the urine, as though somewhere an obstacle to the free passage had been met with. At first only isolated bacilli ap-

peared in the urine, but later they were found in large numbers. In one instance he extirpated one kidney, and two days later, during the first stage of compensatory hypertrophy of the remaining organ, he injected a culture directly into the carotid artery. The animal died suddenly, two and one-half hours after the injection, in an attack of convulsions. Under strict antiseptic precautions the urine was removed from the bladder, and with it a culture of agar-agar was inoculated. The next day the culture showed a beautiful growth of the same bacillus. The author believes that the kidney, the seat of increased vascular pressure, furnished a favorable condition for the rapid passage of the microbe. He found the microbes most frequently in the glomeruli and in the space between these and Bowman's capsule; and again, quite abundant in the blood-vessels and in the lumen of the first portion of the convoluted tubuli uriniferi, and only rarely in the perivascular spaces. Only once was a bacillus found between two epithelial cells of the convoluted tubules. In the cells themselves no bacilli were found.

Seitz found the bacillus of typhoid fever in the urine in two out of seven cases, Konjajeff in three out of twenty cases, Kneppé once in sixteen cases, and Neumann in eight out of forty-eight cases. The last observer ("Ueber Typhus Bacillen im Urin," *Berliner klinische Wochenschrift*, February 10, 1890) in some instances found them so numerous that under the microscope the urine appeared like a fluid culture. In such cases the bacillus multiplies in the bladder. In two cases he also found the streptococcus pyogenes, an occurrence which he considered as an evidence of the existence of complications.

Sittmann (*Deutsches Archiv für klinische Medicin*, September 4, 1894) reports the results of experiments to determine the elimination of staphylococci with the urine. Rabbits were used in the experiments, and were injected in the vein of the ear with staphylococcus pyogenes aureus. After varying intervals the animals were slowly killed with chloroform and cultures made from the arterial and venous blood obtained from the right and left ventricle and from the urine. The microbes were invariably found in the urine. In the severe infection the bacteria appeared after eight hours, in lighter infection after five hours; and this condition was so constant that it was possible to predict the degree of virulence of the infection by the time of appearance of bacteria in the urine.

The elimination ceased, as a rule, after forty-six hours. The author makes the statement that staphylococci can be eliminated with the urine without causing serious damage to the kidney.

Neumann ("Ueber die diagnostische Bedeutung der bakteriologischen Untersuchungen bei inneren Krankheiten," *Berliner klinische Wochenschrift*, 1888, Nos. 7, 8, and 9) found the specific microbes in the urine in cases of typhoid, septicæmia, and pyæmia. In a case of acute endocarditis and acute osteomyelitis, he cultivated from the urine the staphylococcus pyogenes aureus. He believes that the micro-organisms which circulate in the blood localize in the capillary vessels of the kidney, where they often cause minute multiple lesions without implication of the entire parenchyma of the organ. Through the altered tissues some of the microbes enter the tubuli uriniferi and are eliminated with the urine.

The elimination of tubercle bacilli with the urine in phthisical subjects and in animals rendered tubercular by inhalation or inoculation has been proved by many reliable clinical observers and careful experimenters.

Philipowicz ("Ueber das Aufbreiten pathogener Microorganismen im Harne," *Wiener medicinische Blätter*, 1885, No. 22) found the bacillus of tuberculosis in the urine not only in three cases of tubercular pyelonephritis, but also in cases of acute miliary tuberculosis. If the organisms were not present in sufficient number for detection by the microscope, their presence in the urine could be proved by the injection of the urine into the peritoneal cavity of guinea-pigs. He also found bacilli in the urine in cases of glanders. In mice which had died of anthrax the urine contained bacilli in large numbers. In patients who had succumbed to ulcerative endocarditis, pus microbes were found in the urine.

Lentz ("Experimentelle Untersuchungen über die Infektiosität des Blutes und Urines Tuberculöser," Dissertation, Greifswald, 1881) demonstrated the infectiousness of the urine of tubercular persons by inhalation experiments on five rabbits. The animals were confined in a box into which the steam of an atomizer mixed with the urine of two phthisical patients was introduced. For each two of the animals at one sitting thirty to forty cubic centimetres of urine were vaporized. In two of the animals the experiment was continued daily for seventy-one consecutive days. The animals were then killed, and numerous tubercles were found in the lungs,

peribronchial glands, and liver. In three of the animals the urine was allowed to decompose under a cover of filter-paper, when it was administered by the same method. One of the animals died in consequence of an abortion on the ninth day, one was killed on the forty-eighth day, the other on the sixty-ninth day. In all of the animals well-marked tubercular processes were found in the lungs and bronchial glands. The inhalation of urine from healthy persons proved harmless. From the clinical experience and the results of experimentation quoted it is evident that the elimination of pathogenic microbes through healthy kidneys may become a source of bacterial invasion of the bladder sufficient in intensity to produce an inflammation if the microbes find a soil favorable for their reception and growth. This route of infection must be suspected in all cases of cystitis in which more direct infection and infection from the blood can be excluded. The existence of a suppurative affection of the kidneys or pelves, which so often precedes a similar disease of the bladder, greatly enhances the danger of infection by the urine, as the pus microbes multiply in the suppurating focus or foci and reach the bladder by way of the urine in immense numbers, besides the tendency of the infective process to extend from the primary lesion along the ureters to the bladder by continuity of surface.

*Infection from Adjacent Organs.*—The bladder is often involved secondarily by the direct or indirect extension of an infective process from one of the adjacent organs. In females cystitis secondary to suppurative affections of any of the pelvic organs is of frequent occurrence. The bladder may become involved by the direct extension of an infection from its vicinity, or by infection through the lymphatics. In the latter case the inflammation is first interstitial, but may involve the mucous lining by extension of the infective process or by the rupture of an interstitial abscess into the bladder. Extension of a suppurative process in the region of the appendix or the prevesical space in the direction of the bladder is also not infrequently the direct cause of a suppurative cystitis. From an etiological stand-point cystitis caused by the migration of microbes from the intestinal canal is of the greatest importance, and has recently excited a wide-spread interest among bacteriologists and surgeons. The microbe which finds entrance most frequently into the bladder by this route is the bacillus coli commune. The

lymphatics are undoubtedly the channels through which the invasion takes place. This microbe is found so constantly in the intestinal canal, even in healthy persons, and is, according to recent researches, so often found as the sole microbic cause of cystitis, that we are forced to admit that this mode of infection is of frequent occurrence. Clinical data as well as the results of experimentation combined to prove that the bladder is most frequently infected with this microbe from the rectum. The migration of the colon bacillus from the intestinal canal into the bladder under certain conditions has been assumed by Fränkel, Tavel and Lanz, Arnd, Ziegler, and Blom.

Posner and Lewin ("Untersuchungen über die Infektion der Harnwege," *Centralblatt für die Krankheiten der Harn- und Sexual-Organen*, 1896, Band vii., Heft 7 and 8) have furnished experimental proof of the possibility of such an occurrence. Their experiments on animals consisted in first making an intestinal obstruction by closing the anal orifice either by ligature or clamp. After one or two days the urethra was made impermeable in a similar manner. A day or two later the animal was killed and the urine examined. In a number of successful experiments a pure culture of the colon bacillus was found in the urine. These experiments prove that infection of the bladder from the rectum may take place without any gross intestinal lesions. In the same manner colored solutions injected into the rectum found their way into the bladder. Experiments with the bacillus prodigiosus and pyocyaneus yielded the same results. These observers found as the microbic cause of cystitis the bacillus coli, proteus vulgaris, staphylococcus pyogenes aureus and albus, and several times diplococci.

Wreden (*Blätter für klinische Hydrotherapie und verwandte Heilmethoden*, 1895, No. 5), in his experiments on rabbits made for the purpose of demonstrating the possibility of infection of the bladder from the rectum, assured himself first that the urine was free of micro-organisms, then he injured the rectal mucous membrane by injections of very hot water, croton oil, or by irritating it by mechanical means, with the result that cystitis developed, and examination of the urine showed the existence of the same microbes as were found in the rectum. If a tampon containing the proteus or the bacillus mesentericus vulgaris was introduced into the rectum

prepared for infection in a similar manner, the bacilli sooner or later appeared in the bladder.

R. Fultin ("Beiträge zur Frage von den Wegen auf denen *Bacterium coli communis* in die Blase eindringt," Dissertation, Helsingfors, 1896) repeated the experiments of Wreden, and never observed bacteriæmia. In experiments in which the kidneys were damaged by cantharidin the bacillus coli injected directly into veins appeared in the urine. In one series of experiments he injured the rectal mucous membrane with a sharp curette or croton oil, or both, after which carmine-stained bacteria were injected into the rectum. No cystitis resulted and no bacteria could be found in the bladder. In another series of experiments the rectum was treated in the same manner before injecting the microbes, after which the penis was ligated for from twenty-four to twenty-eight hours. In nearly all of these cases the microbes found their way into the bladder in sufficient quantity and virulence to produce cystitis in from three to four days. In a third set of experiments only the penis was ligated. After the removal of the ligature bacteriæmia was found in about one out of five cases. If the animal were catheterized before ligation of the penis, or if the ligature were renewed after two or three days, a cystitis developed almost invariably. In the last experiments the rectum was seriously injured before the bacterial injection was made. In some of the experiments bacteria entered the bladder and produced cystitis, but peritonitis was also observed with about the same degree of frequency. In these cases bacteria were also found in the blood and kidneys. If the animals survived, the urine remained sterile, except in one instance, in which a cystitis developed slowly after a few days. In drawing conclusions from the results of his experiments, he admits that in some instances the bladder may become infected from the rectum, but he maintains that this mode of infection is comparatively rare and not as constant as claimed by Wreden. Clinical observations fully corroborate what has been established by experiments on animals,—namely, that cystitis is quite frequently caused by infection from the rectum, and that this method of infection is very likely to take place when lesions of the mucous membrane of the rectum are present, which admit of a more ready entrance into the tissues and migration of the bacillus coli, and when the bladder, from retention of urine or

antecedent injury or disease, is in a condition of increased susceptibility to infection.

*Infection from the Blood.*—In rare cases infection of the bladder takes place by microbes which float in the general circulation with or without an antecedent or coexisting suppurating depôt in some other part of the body. It has become an established fact, in connection with the origin of many of the surgical infective lesions, that the blood in the general circulation contains pathogenic microbes which produce no ill results until some tissue or part of the body becomes prepared for their localization and growth, when infection takes place in the prepared soil. Such direct connection between cause and effect has been repeatedly demonstrated experimentally between pyogenic microbes in the general circulation and the causation of suppurative osteomyelitis, synovitis, peritonitis, and tuberculosis of bones and joints. It is only rational to assume that in rare cases cystitis is produced by this method of invasion. We should naturally expect that in such cases the infection begins beneath the mucous membrane or in the middle coat, and that the lesion of the mucous membrane resulting as a secondary consequence begins as a circumscribed affection and often remains so unless retention of urine sets in and favors the dissemination of the infection over the surface of the mucous lining of the bladder. Ulcerative cystitis unquestionably has occasionally such an origin. In other cases pyogenic microbes are eliminated in sufficient number through the intact kidneys and reach the bladder with the urine, retaining a sufficient degree of virulence, when brought in contact with a favorable soil, to induce an attack of cystitis. Usually under such circumstances the cystitis is of a diffuse character. Retention of urine in such instances constitutes the most frequent predisposing cause. Infection from the blood in the causation of cystitis merits further experimental study, which will undoubtedly throw more important light upon the etiology of this disease. In recapitulating what is known of the causes of cystitis, I am warranted in stating that a healthy bladder containing normal urine is seldom attacked by inflammation, and that when inflammation of this organ does occur, it is in consequence of the presence of one or more of the predisposing causes which have been enumerated and the entrance into the bladder through one of the routes mentioned of pathogenic microbes in sufficient number and virulence to produce inflamma-

tion in the tissues prepared for their pathogenic action by antecedent or co-existing injuries or disease.

## II. CLASSIFICATION OF CYSTITIS.

A rational classification of cystitis is essential in discussing the etiology, symptomatology, diagnosis, prognosis, and treatment of this disease. The surgeon is no longer content to simply recognize the existence of the disease. To enable him to estimate the gravity of the affection and to adopt an intelligent course of treatment, he must be in possession of an accurate knowledge of its real nature, location, and extent. He must know what microbe or microbes have produced the inflammation before he can make a diagnosis that will suggest the necessary therapeutic indications. Mistaken and inaccurate diagnoses are largely responsible for the many short-comings of our present therapeutic resources. For the purpose of showing some of the defects of the methods of classification of cystitis heretofore made by authors who have made a special study of this disease, I will give only the classification made by Guyon and Rovsing.

*Guyon's Classification of Cystitis.*—1, Cystite blennorrhagique; 2, cystite tuberculeuse; 3, cystite calculeuse; 4, cystite des rétrécis; 5, cystite des prostatiques; 6, cystite des néoplasiques; 7, cystite chez la femme; 8, cystite douloureuse; 9, cystite membraneuse.

I can see no reason for discussing cystitis as it occurs in women as a separate affection; neither is it necessary in classifying cystitis to consider separately the different causes which give rise to irritation of the bladder or retention of urine. Cystitis is always a painful affection, and there is absolutely no excuse for setting aside a class of cases in which this symptom may be more prominent than in others as a separate variety under the head of cystitis douloureuse.

*Rovsing's Classification of Cystitis.*—1, Cystitis catarrhalis; 2, cystitis suppurativa. The latter class he subdivides again into (a) cystitis suppurativa ammoniacalis and (b) cystitis suppurativa acidula tuberculosa.

Rovsing's classification has special reference to the action of the essential microbic cause on the urine and the tissues of the bladder, but it is well known that in many cases of non-tubercular cystitis the urine is acid, especially in cases in which the colon bacillus is found as the sole microbic cause. This classification also ignores



almost completely the different pathological varieties with which the surgeon must be familiar in order to comprehend the nature and extent of the disease. No classification is complete which does not indicate the anatomical location, the clinical features, pathological characteristics, and bacteriological origin of the disease.

1. *Anatomical Classification*.—(a) Pericystitis; (b) paracystitis; (c) interstitial cystitis; (d) endocystitis.

(a) *Pericystitis*.—Cystitis is the term usually employed in designating an inflammation of the bladder without any special reference to what tissues are the seat of the inflammatory process. In the diagnosis of all diseases it is of the greatest importance to determine, if possible, the organ or tissues in which the disease had its starting-point; in other words, to make first an anatomical diagnosis. Although the mucous membrane of the bladder is most frequently primarily affected in cystitis, either of the remaining two tunics may be the primary starting-point of the inflammatory process from which the infection may or may not extend to the mucous membrane, but the symptoms usually point in that direction.

Guyon ("Diagnostic différentiel de certaines formes de la cystite et des néoplasmes de la vessie," *Annales des Maladies des Organes Génito-urinaires*, 1895, No. 4) calls attention to the great difficulties which the surgeon frequently encounters in the differential diagnosis between cystitis and neoplasms of the bladder. He demonstrated in his clinic two patients in which, besides the usual symptoms of cystitis, an infiltration of the bladder wall simulated tumor. Suprapubic incision of the bladder cleared up the diagnosis, as it revealed a well-marked pericystitic infiltration. In a third case the symptoms which had existed for eight months appeared to point directly to cystitis, when a perineal section made it clear that it was a case of carcinoma.

In pericystitis the peritoneal coat of the bladder is the seat of inflammation; it is in reality a vesical peritonitis. This anatomical form of cystitis follows usually in consequence of the extension of an inflammation from one of the adjacent organs,—the appendix, uterus, tubes, or ovaries; in rare instances it is caused by intestinal perforation. The inflammatory product is most abundant around the base and at the sides of the bladder. At the sides of the bladder, at a point corresponding with the vesical end of the ureter, the inflammatory masses often reach considerable size, and by cicatricial

contraction may eventually cause ureteral obstruction. In the female, vesical peritonitis is usually secondary to pelvic peritonitis. The immobilization of the bladder by adhesions and the vascular disturbances caused by the pericystitis are often productive of great vesical distress, and secondary pathological changes often reach the mucous membrane of the bladder.

Dacheux (*Centralblatt für Gynäkologie*, 1895, No. 40) believes that the condition of irritable bladder in women, which has previously been regarded as a purely functional disorder, is really, as Zuckerkandl states, due to localized hyperæmia of the mucous membrane, which can be demonstrated by cystoscopic examination. Hyperæmic patches are seen at the base of the bladder, and less often at the neck, which bear a close etiological relation to concomitant congestion of the uterus and adnexa, and often disappear when the latter is relieved.

Kolischer (*Centralblatt für Gynäkologie*, 1895, No. 27) describes, in connection with such cases, a peculiar form of oedema of the mucous membrane of the bladder observed through the cystoscope. It appears in the form of circumscribed blisters the size of a pea, the rest of the membrane being normal. In some cases the blebs are so large as to resemble vesicular moles. This pathological condition is always associated with pelvic exudates near the bladder, and is seen most frequently in women the subjects of salpingitis. The symptoms which attend this form of vesical irritation are painful urination, tenesmus, and a feeling of weight and pressure over the bladder. While the clinical symptoms indicate the presence of cystitis, examination of the urine yields negative results, unless the infection has extended to the mucous membrane of the bladder.

(b) *Paracystitis*.—Paracystitis is an inflammation of the subperitoneal connective tissue of the bladder or of the surrounding connective tissue where the bladder is extraperitoneal in the cavum Retzii and prostatic portion of the organ. At the base of the bladder a paracystitis not infrequently develops in the course of a suppurative prostatitis, and in front of the bladder the disease usually appears in the form of a phlegmonous inflammation of the loose connective tissue in the cavity of Retzius. In both of these locations abscess formation is the usual termination of the inflammatory process, an occurrence always attended by distressing bladder symptoms. Abscesses in both of these places, unless incised early, are

very prone to rupture into the bladder, an accident which is often followed by an obstinate cystitis. Inflammation of the subserous connective tissue, following infection through the lymphatic channels, is a very obscure affection, and a positive diagnosis is more frequently made in the post-mortem room than at the bedside. The formation of multiple abscesses in such cases is not an unusual occurrence. More or less pericystitis is almost always associated with pericystitis involving the intraperitoneal portion of the bladder.

(c) *Interstitial Cystitis*.—Interstitial cystitis implicates the middle tunic or muscular coat of the bladder. It is in this coat that we find the lymph channels most numerous and consequently the most common pathways of infection. Invasion of the middle coat may take place from either direction, either by extension of infection from the mucous lining, or the microbes may reach the muscular coat through the lymph-channels from some adjacent inflammatory focus. Another, but perhaps the rarest, route of infection is through the circulation, when the interstitial cystitis is initiated by an endo- or perivascular inflammation. Direct extension of inflammation of the mucous membrane to the muscular coat, or invasion by way of the lymphatic channels, usually leads to a diffuse inflammation in which the whole middle coat may participate, in which case it becomes greatly thickened and the mucous membrane is changed into folds. Ridges can be felt with the sound. Interstitial cystitis usually leads to abscess formation. Small abscesses develop in the submucous connective tissue, or in the muscular coat, which, when they open into the bladder, leave diverticula which heal slowly and in which calculi are often found concealed. Recovery from this form of cystitis often results in a great diminution in the size of the bladder, caused by cicatricial contraction. In the embolic form of interstitial cystitis circumscribed inflammation and abscess formation are the usual results of the infection.

(d) *Endocystitis*.—What is ordinarily understood by cystitis is an inflammation of the lining membrane of the bladder. The inflammation may almost from the beginning, or at any rate in a few days, involve the entire surface of the mucous membrane, or it may remain limited to certain localities which are most exposed to infection. Localized cystitis is found most frequently in the trigonum and about the urethral and ureteral orifices; it is also from these points that diffuse cystitis has its starting-point. The neck

of the bladder is the most sensitive part of the organ, and it is here that inflammation gives rise to the most distressing symptoms. In cystitis proper the urine contains, almost from the very beginning, the morphological elements of the inflammatory product,—blood, epithelial cells, and pus-corpuscles,—the presence of which always constitutes an important distinguishing feature between endocystitis and the other anatomical varieties of inflammation of the bladder. In the further discussion of the classification of cystitis, endocystitis, or cystitis proper, will be taken as the type of the disease.

2. *Pathological Classification.*—(a) Catarrhal cystitis; (b) suppurative cystitis; (c) ulcerative cystitis; (d) exudative cystitis; (e) exfoliative cystitis.

The effects of microbes and their toxins on the tissues of the bladder vary according to the specific pathogenic effect of the original bacterial cause and the number of microbes and their degree of virulence. The inflammatory product is also greatly influenced by the condition of the urine and the nature and extent of the predisposing causes. The successful treatment of cystitis is often materially influenced by the character of the inflammation and the nature of complications which may precede, attend, or follow cystitis. I am sure every surgeon will appreciate the value and importance of a clear pathological classification of cystitis for the purpose of grouping his cases properly from a pathological standpoint and as a reliable aid to diagnosis and treatment. The pathological classification must be based entirely on the character of the inflammatory product.

(a) *Catarrhal Cystitis.*—For a long time catarrh of the bladder has been regarded as synonymous with chronic inflammation. German writers have been particularly partial to this term, which has made so much confusion in the literature on inflammatory affections of the bladder. From a modern pathological stand-point catarrhal cystitis is a term used to indicate the existence of a superficial inflammation of the interior of the bladder in which the epithelial cells furnish the principal part of the morphological elements of the inflammatory product. It is, like all catarrhal inflammations in other localities, a surface affection. The mucous membrane is swollen, red, and the inflammatory process consists in increased exfoliation of epithelial cells and an abundance of mucus formation. If the disease becomes chronic, thickening of the mucous membrane

and secondary infiltration of the muscular coat lead to hypertrophy of the bladder wall. Retention of urine aggravates the inflammation and increases the vesical distress. Erosions and superficial ulcerations may develop during the course of the disease. The urine is usually acid, and contains pus and an abundance of bladder epithelium. In cases in which the urine has undergone alkaline decomposition the inflamed surface presents a dirty-whitish deposit of muco-pus.

Rovsing ("Om Blaerebetaendelsernes Aetiologi, Pathogenese og Behandling," Kjoebenhavn, 1889) is of the opinion that in catarrhal cystitis the microbes which produced the disease do not attack the mucous membrane of the bladder, but that they provoke the disease by rendering the urine ammoniacal, while in suppurative cystitis, in addition to this, their pyogenic action is expended upon the mucous membrane prepared for their specific action on the tissues by the preceding catarrhal inflammation. It is not always easy and sometimes impossible to draw a sharp line between catarrhal cystitis and suppurative cystitis because the latter frequently follows upon the footsteps of the former, and the transition is often so gradual that it is impossible to tell where one ends and the other begins. From a clinical stand-point the differentiation between these closely allied inflammatory affections of the bladder is perhaps attended by less difficulty, as suppurative cystitis gives rise to more serious constitutional disturbances than catarrhal cystitis, owing to the existence of a more intense infection and more extensive involvement and destruction of tissue.

(b) *Suppurative Cystitis*.—Suppurative cystitis appears clinically usually as a diffuse affection, in which not only the epithelial lining but also the deeper structures are generally involved. The microbic infection is of sufficient intensity to destroy the protoplasm of the morphological products of the inflammation, white corpuscles purely epithelial, and connective-tissue cells, and transform them into pus-corpuscles. The urine contains large quantities of pus and bladder epithelium. During the acute stage small fibrinous patches appear upon the inflamed surface. Ulceration differing in extent and depth is of common occurrence. Deep necrosis may lead to perforation. If the urine is ammoniacal, the necrosed patches present a grayish-white color and are encrusted with sand-like deposits. The decomposition of the urine is generally due to other microbes

than those which have caused the suppurative inflammation; that is, it is generally the result of a mixed infection. Besides the usual pyogenic microbes, the ammoniacal urine contains some species of saprophytic bacteria or the diplococcus ureæ. Suppurative cystitis generally begins as an acute inflammation, but is very liable to pass into the chronic form, and direct extension of the infective process is liable, sooner or later, to implicate the kidneys.

(c) *Ulcerative Cystitis*.—In this class of cystitis it is not my intention to include the cases of suppurative cystitis which terminate in ulceration which would only indicate an advanced stage of the disease, but I desire to limit the application of the qualifying term ulcerative to a form of cystitis in which ulceration takes place almost from the beginning of the inflammation. In cases of this kind the infection appears to be of a peculiar kind, limited in extent, and the resulting inflammation leads quickly to a circumscribed destruction of tissue, the formation usually of a single circumscribed ulcer, the so-called “simple” ulcer of the bladder. This form of cystitis is quite rare, and resembles in many respects gastric ulcer and the round duodenal ulcer.

Fenwick (*British Medical Journal*, May 9, 1896) has seen a number of cases of simple, solitary ulcer of the bladder. The disease is usually met with in young men without a venereal history. The first symptom is increased desire to urinate, coming on suddenly; intermittent hæmaturia then appears. He describes three stages. In the first stage the urine is acid, the specific gravity high, and pus scanty. In the second stage the ulcer becomes encrusted with phosphates, and fragments of the deposit break off now and then and are passed with painful paroxysms, or are retained and serve as nuclei for calculus formation. During the third stage the bladder becomes contracted, the mucous membrane extensively ulcerated, and ureteral and renal lesions arise. Solitary ulcer is clinically indicated if the urine is clear and normal in specific gravity and reaction, and if there is constant penile pain and absence of nocturnal irritability. This form of cystitis is undoubtedly the result of an infection from the blood, the inflammation attacking the tissues around an infected embolic infarct, reaching the surface of the bladder by a process of ulceration. Like gastric and duodenal ulcer, ulcerative cystitis is found, as a rule, in young adults,

and usually without any antecedent or attending predisposing local causes.

(d) *Exudative Cystitis*.—Inflammation of the mucous membrane of the bladder accompanied by the deposition upon the inflamed surface of the products of coagulation necrosis should be called exudative cystitis. The descriptive terms, membranous, diphtheritic, croupous, and fibrinous, are confusing and misleading and should be excluded from the present nomenclature in the description of this pathological form of cystitis. The exudate consists largely of fibrin, and is variously modified in quantity and appearance by the character of the infection and the condition of the urine. The exudate is the best possible proof of the severity of the infection and intensity of the inflammation. It proves the existence of a deep-seated lesion and great damage to the blood-vessels in the inflamed tissues. This form of cystitis is most frequently observed in puerperal women and women suffering from pelvic tumors large enough to subject the bladder to harmful pressure.

According to Stein, of fifty cases of exudative cystitis, forty-five occurred in women mostly in connection with the puerperal state or tumors of the uterus. In the milder types of this disease the mucous membrane under the fibrinous exudate is vascular and swollen; in the grave cases the mucous membrane and submucous connective tissue are often extensively destroyed by acute necrosis or ulceration.

Adami (*Montreal Medical Journal*, July, 1894) studied a case of this kind in a woman the subject of a myofibroma of the uterus large enough to produce considerable pelvic pressure. To pressure and obstruction of the lymph-stream he attributed important elements in the etiology of this variety of cystitis. The membranes and shreds were passed after great pain and distress. The membranes were found to be composed of a large amount of fibrin, and incorporated in this what were evidently layers of the bladder wall. In many of the cases not only epithelial layers but a certain amount of the muscle tissue of the bladder wall has thus become destroyed.

Savor ("Cystitis crouposa bei saurem Harn," *Wiener klinische Wochenschrift*, 1895, No. 44) observed a case of exudative cystitis on the fourth day after extirpation of the uterus by the abdominal route. Catheter was not used either before or after operation. Membranes five to ten centimetres in length were expelled with the urine. These membranes were composed of fibrin and contained in

the meshes of the fibrin numerous pus-corpuscles. The urine was ammoniacal only for one day. In the urine sediment the colon bacillus was found, and was regarded by the author as the essential microbic cause of the inflammation. Savor made experiments with pure cultures of this bacillus with a view of reproducing this special form of inflammation upon serous and mucous surfaces of other organs in animals, but the results of his endeavors proved negative. The urine in exudative cystitis is usually alkaline, and Savor believed that in his case it remained acid after the first days owing to the absence of a mixed infection. In the majority of cases exudative cystitis occurs in women during the childbearing period and positive proof of the pathological nature of the cystitis is always furnished by the expulsion of membranes or shreds of fibrin with the urine.

(e) *Exfoliative Cystitis*.—Exfoliative cystitis is an inflammation of the bladder in which, almost from the very beginning, the toxins of the microbes which produced the disease destroy the mucous membrane and sometimes even the muscular coat, which, if the patient survive, become detached with the inflammatory product and are expelled with the urine, or in some instances have to be extracted from the bladder by the surgeon. This is the most dangerous form of cystitis, and can only occur as the result of a most virulent infection, aided in most cases by local predisposing causes. In exudative cystitis the toxins precipitate the inflammatory product by causing coagulation necrosis; in exfoliative cystitis they cause necrosis of the mucous lining of the bladder and occasionally also of the muscular coat. The same mechanical causes which are so influential in causing exudative cystitis are usually present and active in the production of exfoliative cystitis. In a few cases carcinoma of the bladder appears to have been the predisposing cause of this form of cystitis, with the result that the disease was completely removed by the extensive sloughing, and in a few instances the patients not only recovered from the cystitis, but were permanently cured of the carcinoma by the complete elimination of the malignant tissues with the product of the exfoliative process.

A very interesting case of extensive exfoliative cystitis is reported by J. C. Warren (*Boston Medical and Surgical Journal*, June 25, 1896). Under an anæsthetic "rolls of sloughing tissue" were extracted with forceps until a "membrane the size of a small pocket-handkerchief" had been removed from the bladder in a con-



nected mass. The patient suffered first from incontinence, but gradually recovered under daily washings out of the bladder with a weak solution of potassium permanganate, and later with a solution of boracic acid. An examination of the specimen removed showed it to be of a character similar to the submucosa of the bladder.

Boldt (*American Journal of Obstetrics*, April 27, 1889), under the term suppurative exfoliative cystitis, describes this form of cystitis, marked by inflammatory separation *en masse* of portions of the mucous lining of the bladder, due especially, as he believes, to posterior displacements of the gravid uterus, to undue or prolonged pressure of the foetal head in labor, or to other mechanical causes. The degree and extent of the exfoliation vary with the intensity of the mechanical cause and the virulence of the infection. The differentiation between exudative and exfoliative cystitis can only be made by a careful study of the membranes, shreds, or masses expelled or removed from the bladder, which often must necessarily include the use of the microscope as a diagnostic aid.

3. *Clinical Classification*.—(a) Acute cystitis; (b) chronic cystitis.

(a) *Acute Cystitis*.—The old and usual clinical diagnosis is based on the intensity of symptoms and duration of the disease. That form of inflammation of the bladder in which the symptoms appear suddenly and reach their maximum height in a short time is known as acute cystitis. In this class of cases the infection is intense, the constitutional disturbances well-marked, and the nature of the pathological products in accordance with the acuity of the inflammatory process. One of the best illustrations of what is meant by acute cystitis is furnished by cases of urine retention in which infection occurs by the use of the catheter. The disease is usually initiated by a chill followed by febrile reaction; the urine becomes turbid within twenty-four hours, and in a few days contains large quantities of pus, and ammoniacal decomposition is developed very rapidly. It is not difficult in the majority of cases to establish the existence of acute cystitis, but such a diagnosis no longer satisfies the surgeon who seeks to complete his diagnostic work by investigating the pathological anatomy of the disease and by ascertaining the nature of the infection. Under appropriate treatment an acute cystitis may be under control in a short time, but in the presence of obstructive or visceral lesions the acute symptoms subside in the

course of time, when the disease only too often passes into the chronic form.

(b) *Chronic Cystitis*.—Chronic inflammation of the bladder is characterized by the absence of acute symptoms, local and general, and the tendency of the disease to persist regardless of the treatment employed. The suppurative form of chronic cystitis is usually complicated by the coexistence of stricture of the urethra, enlarged prostate, or the presence of stone or foreign body in the bladder. Cystitis caused by infection from a suppurative affection of the kidneys is also very prone to pursue a chronic course, as the constant irrigation of the bladder with infected pus maintains an uninterrupted source of infection. The best example illustrating the clinical aspects of chronic cystitis is furnished by the tubercular variety. The disease begins insidiously by the appearance of isolated symptoms which point to the bladder as the probable seat of the inflammation. The symptoms gradually increase in number and intensity until the complexus is complete upon which to base a diagnosis of chronic cystitis. The symptoms are often masked by complications which served as predisposing causes or which ensued in consequence of the chronic inflammation. It is in cases of chronic cystitis that an early and correct diagnosis is so seldom made. Renal disease is often mistaken for cystitis, and cystitis for renal disease. It is in such cases that a recourse to all modern diagnostic aids is indispensable for a correct interpretation of the symptoms as they arise. It is not unusual that patients suffering from incipient tuberculosis of the bladder go from one physician to another, and are repeatedly sounded for stone in the bladder, when perhaps the results obtained from a careful examination of the external genitals and the prostate would at once arouse suspicions in reference to the probable tubercular nature of the vesical affection. It is well to remember that in the majority of cases of chronic inflammation of the bladder not complicated by obstructive lesions the disease is of a tubercular nature.

4. *Bacteriological Classification*.—(a) *Bacillus coli commune* infection; (b) saprophytic (mixed) infection; (c) *staphylococcus* infection; (d) *streptococcus* infection; (e) *streptococcus erysipielatis* infection; (f) *typhoid bacillus* infection; (g) *diplobacillus* infection; (h) *gonococcus* infection; (i) *bacillus of tuberculosis* infection.

The bacteriological classification of cystitis is the most modern

and certainly the most important. The bacteriological classification has a direct bearing on the etiology of the disease, and suggests to the surgeon the most rational course to pursue in its treatment. In long-standing and obscure cases of inflammation of the bladder examination is not complete without an examination of the urine with sufficient care and thoroughness upon which to base a correct bacteriological classification. Surgeons must learn to appreciate the value and importance of this part of the examination before we can expect material advances in the treatment of cystitis. If the surgeon has not the necessary knowledge or equipment to make these examinations satisfactorily, he should assign this part of his task to a competent bacteriologist.

(a) *Bacillus Coli Commune*.—Bacteriological researches made by numerous observers go to prove that the bacillus coli commune is found more frequently in the urine of patients suffering from cystitis than any other known microbe, and all combine in assigning to it a more or less important rôle in the causation of the disease. This micro-organism was discovered by Escherich in 1886 ("Die Darmbakterien des Säuglings," Stuttgart, 1886). It is constantly found in the contents of the normal intestinal canal. Its presence in suppurative affections in different parts of the body has been repeatedly demonstrated, and its pathogenic qualities have been carefully determined. Since 1891 the colon bacillus has been demonstrated in the urine in cystitis patients by Achard and Renault, Reblaub, Haushalter, Hartmann, Guinon, and Denys, all of whom claim for it distinct etiologic qualities.

Rovsing ("Die Bedeutung des Bacterium Coli für die Pathologie der Harnorgane," Hospitals Tidende, No. 32, 1895) has reported a number of cases of infection of the upper portion of the urinary tract in which myriads of the colon bacillus were found in the urine without having produced cystitis. If in such cases another microbe finds its way into the bladder which will decompose the urine, cystitis invariably follows. The colon bacillus does not decompose urea. The ordinary pus microbes decompose the urine, and are almost constantly found in cases of cystitis in which the colon bacillus is found in abundance. The microbes which are known to decompose urea are bacterium pyocyaneus, cocci bacillus pyogenes ureæ, staphylococcus, and a special form of streptococcus found in decomposed urine.

Rovsing ascertained, also, that in dead culture media the colon bacillus exerts a destructive, or at least an inhibiting effect on the bacteria which decompose urine. The correctness of these observations is sustained by the investigations of Beco, Wotholet, Charin and Veillon, Tavel and Lanz, Albarran, Halle, Melchior, Guyon, Schmidt, Krogius, and Schoff, all assigning to the bacillus coli distinct and important pathogenic properties in the genesis of cystitis. Recent observations tend to prove that the colon bacillus requires the co-operation of urine-decomposing bacteria in the production of cystitis.

Trumpp ("Ueber Colicystitis in Kindesalter," *Münchener medicinische Wochenschrift*, 1896, No. 42) made a bacteriological examination of the urine in twenty-nine cases of cystitis in children (eight boys, twenty-one girls), in the clinic of Escherich. Of these cases, seventeen were suffering at the same time from follicular enteritis. In fourteen the bacillus coli was found. The cystitis in these cases was characterized by grave general symptoms, slow progression of the infective process in the direction of the kidneys, and occasionally a fatal nephritis.

Melchior found the urine acid in all cases of cystitis in which the colon bacillus was found as a solitary microbe. His experiments on rabbits, guinea-pigs, and mice with pure cultures injected into veins, beneath the skin, and into serous cavities proved the pyogenic properties of this microbe. He is of the opinion that many of the bacilli discovered in the urine of cystitis patients and described under different names are identical with the colon bacillus. Of thirty-seven cases of cystitis examined by Melchior, the colon bacillus was found in thirteen (twelve times solitary); diplococcus ureæ liquefaciens, eleven times (nine times solitary); proteus Hauser, five times (three times solitary); and staphylococcus pyogenes, four times (three times solitary). It seems that the colon bacillus under certain favorable circumstances can and does produce cystitis, but it is equally certain that its pathogenic properties are greatly enhanced when it is associated with bacteria which decompose the urine. It is found most frequently in the urine in cases of cystitis caused by a mixed infection. Colon bacillus infection should be suspected in all cases of cystitis following operations upon the rectum, and when the disease has followed or is coexistent with intestinal fermentation, irritation, or inflammation; also in cases in which the urine remains acid.

(b) *Saprophytic (Mixed) Infection*.—In more than one-half of all cases of acute and chronic cystitis, infection is the result of the presence and combined action of several different kinds of microbes. Pus microbes and the saphrophytes decompose the urine, rendering it alkaline. Ammoniacal urine acts as an irritant to the mucous membrane of the bladder, producing textural changes which prepare the way for the action of the bacteria, which are more directly concerned in the production of the inflammation. A mixed infection must be suspected in all cases in which the urine is ammoniacal.

Mesnil de Rochemont (*op. cit.*) cultivated from the urine of twenty-five cases of cystitis fourteen different kinds of microbes, one-half of which decomposed urea; the remaining half had no such effect.

Kastalskaya (*Zeitschrift für Geburtshülfe und Gynäkologie*, Band xxxv., Heft 1), from a bacteriological study of urine from the bladder in twelve cases of cystitis, reports the following results: In one case he found the bacillus foetidus liquefaciens mixed with tubercle bacilli and non-pathogenic cocci; in five cases tubercle bacilli, twice as a pure culture, once mixed with non-pathogenic cocci and bacillus coli, and once with non-pathogenic cocci and the bacillus foetidus liquefaciens; in four cases the bacillus coli, twice as a pure culture, once with non-pathogenic cocci and tubercle bacilli, and once with streptococcus pyogenes; in one case pseudobacillus coli commune as a pure culture; in one case bacilli with non-pathogenic cocci; in one case streptococcus pyogenes with bacillus coli; and in one case a pure culture of pseudostaphylococcus albus.

Lépine and Roux, of Lyons (*Comptes-rendus hebdomadaire des Séances de l'Académie des Sciences*, Paris, July, 1894) proved by numerous experiments with dogs and guinea-pigs that the introduction into the urethra of a pure culture of the micrococcus ureæ, followed by temporary ligation of the urethra, invariably resulted in ammoniacal fermentation of the urine and cystitis with grave lesions. Decomposition of the urine occasionally is attended by gas formation (pneumaturia), and in such instances some form of gasogenic bacteria can be found. Heyse (*Zeitschrift für klinische Medicin*, Band xxiv.) records a case of gas formation in the bladder of a patient suffering from myelitis with retention of urine. After catheterization had been practised for some time gas was noticed escaping through the catheter. Autopsy showed gas in considerable

quantities in the bladder, ureter, and substance of the kidneys. Cultures revealed the presence of a short bacillus resembling the bacterium *lactis aerogenes* of Escherich. Pure cultures injected into the tissues of animals always resulted in gas formation.

Schnitzler (*Internationale klinische Rundschau*, February 25, 1894) reports an analogous case in a woman forty-six years old, the only difference being that the catheter had never been used. Here, also, the formation of gas was due to the presence of the bacillus *lactis aerogenes*. The author made experiments with the bacillus *coli* for the purpose of showing its gas-producing effect, but only succeeded in animals he had first rendered diabetic by phloridzin. In the same way the injection of the bacillus *lactis aerogenes* produced violent cystitis, but developed gas only in animals rendered diabetic. These experiments show that decomposition of urea and putrefaction caused by microbes is often greatly influenced by the composition of the urine. Saprophytic infection is almost always associated with urine retention, and may precede or follow infection with the microbes which are the essential cause of the suppurative cystitis. It is in such cases that careful, systematic catheterization and antiseptic irrigations of the bladder prove of such eminent value in correcting the alkalinity of the urine and in arresting the suppurative inflammation.

(c) *Staphylococcus Infection*.—The staphylococcus *pyogenes* *albus* and *aureus*, the microbe most frequently found in all suppurative affections has been often demonstrated as a solitary microbe, and in association with other pyogenic microbes and saprophytic bacteria in the urine of patients suffering from catarrhal and suppurative cystitis. The staphylococcus is a comparatively mild microbe, and its presence as a sole microbic cause should be suspected in inflammatory affections of the bladder in which the infection does not penetrate deeply, and in which the urine shows no evidences of exfoliation. In staphylococcus infection the urine may be ammoniacal without the presence of saprophytic bacteria, as pus microbes, when present in large numbers, decompose the urea, besides lighting up the suppurative inflammation.

(d) *Streptococcus Infection*.—That the streptococcus *pyogenes* is not often the cause of cystitis becomes apparent from a bacteriological examination of the urine from six cases of inflammation of the bladder made by Huber (*Correspondenzblatt für Schweizer*

*Aerzte*, October, 1893). He found this microbe only once; in the remaining five cases the bacillus coli commune.

Melchior found the urine acid in all cases of cystitis in which the streptococcus was found as a solitary microbe. It is well known that the streptococcus generally produces a diffuse form of inflammation, during which the connective tissue is often destroyed by the toxins and is later eliminated or removed in the form of shreds. The streptococcus invades the lymphatic channels and connective tissue spaces, and is almost constantly found in phlegmonous inflammations and diffuse abscesses. A streptococcus cystitis is characterized by the intensity of the local and general symptoms and by more or less destruction of the tissues of the bladder wall. The presence of this microbe may be surmised in cases of diffuse interstitial and exfoliative cystitis.

(e) *Erysipelatous Cystitis*.—Erysipelatous inflammation, either as a primary affection or as a metastatic process, has been found in many of the internal organs. Infection of the bladder with the streptococcus erysipelatis is extremely rare, but there can be no doubt of the possibility of such an occurrence.

Fritsch (*Centralblatt für Chirurgie*, 1894, No. 7) reports the case of a man, aged fifty-two, suffering for a long time from symptoms of suppurative prostatitis, and who was attacked with chills, fever, vomiting, and frequent desire to urinate; the urine, very turbid, contained streptococci, as did also the mucopurulent secretions of the prostate. The erysipelatous character of the cystitis was demonstrated by culture and inoculation experiments. The patient suffered subsequently from an erysipelas of the left thigh, which extended to the right thigh, the back, etc., but eventually he recovered.

(f) *Typhoid Infection*.—Our distinguished member, Professor W. W. Keen, has recently made a most important addition to the literature pertaining to typhoid infection and complications in his classical book on "The Surgery of Typhoid Fever." He mentions a number of cases of typhoid infection of the male and female genital organs, but nothing is said of cystitis as a possible complication or remote consequence of typhoid fever.

Melchior ("Om Cystitis og Urininfektion," etc.) reported the first and probably the only case of cystitis caused by infection with the typhoid bacillus. The patient had been convalescent from an

attack of typical typhoid fever for fourteen days, and had been out of bed for three days, when he was attacked suddenly with a severe form of cystitis, for which there appeared to be no cause, as the catheter had never been used. For several days the urine was sterile, and contained a large amount of pus and some blood. The typhoid bacillus was cultivated from the urine, and was the only microbe it contained. In rabbits injections of pure cultures into the bladder obstructed for eighteen hours by ligation of the penis caused a severe cystitis. Intravenous injections caused death of the animals from typhoid fever with the characteristic anatomical lesions.

(g) *Diplobacillus Infection*.—The diplobacillus of Friedländer, which has been found in so many suppurative lesions complicating or following pneumonia, has in rare instances been found as the only microbic cause of cystitis. In a case of general infection with this microbe which resulted in death, Brunner (*Münchener medicinische Wochenschrift*, Nos. 13, 14, 1896) found at the post-mortem, besides other multiple lesions, hemorrhagic lesions.

Montt-Saverdo ("Zwei Fälle von Cystitis mit Befund von Diplobacillus," Friedländer, *Centralblatt für Bacteriologie*, etc., Band xx., Nos. 4, 5, 1896) found the diplobacillus of Friedländer in the urine of two cases of cystitis, which, in the absence of other microbes, was regarded by him as the essential cause of the disease. The first patient was a man fifty-three years of age who three years previously suffered from an attack of pneumonia. For two months he had been the subject of cystitis characterized by the escape of gas from the urethra. The urine contained pus, small quantity of albumen and diplobacilli, but no gonococci. In the second case the suppurative cystitis was complicated by hypertrophy of the prostate. The symptoms yielded to washings out of the bladder with solutions of salol and nitrate of silver. Five years later there was a return of the cystitis, with phlebitis of the saphenous vein and death, preceded by septic manifestations. The autopsy revealed carcinoma of the prostate and conditions indicated by the clinical course of the disease, also a few bronchopneumonic foci. Bacteriological examination of the urine resulted in the finding of staphylococcus pyogenes and the diplobacillus of Friedländer.

(h) *Gonococcus Infection*.—It has been said that the gonococcus never causes cystitis, and that a gonorrhœal urethritis, as long as it



remains as an unmixed infection, does not extend to the bladder. It is claimed that when cystitis develops in the course of a specific urethritis it is the result of a mixed infection. This is undoubtedly true in the majority of cases, but occasionally a gonorrhœal cystitis is met with in which the gonococcus is found as a solitary microbe in the urine. It is not always easy to demonstrate the gonococcus in the urine in gonorrhœal cystitis. The reason why gonococci are not always found in the chronic cases, as Wertheim has pointed out, is that, while young gonococci are well stained with aqueous solutions of anilin colors, the old germs are pale and imperfectly defined. Moreover, gonococci assume "involution forms," becoming granular and of irregular form when the culture medium becomes old. These forms are not recognizable as gonococci, yet they can be regenerated to the classical form through a fresh culture medium. Gonorrhœal cystitis presents itself more frequently in the form of a localized than a diffuse inflammation of the bladder. The favorite location for this form of cystitis is the trigonum of the bladder. If the disease becomes more diffuse, it manifests a tendency to extend in the direction of the kidneys. The most conclusive proof of the existence of gonorrhœal cystitis has been furnished by Wertheim ("Ueber Blasengonorrhöa," *Zeitschrift für Geburtshilfe*, Band xxxv., Heft 1). In a girl nine years of age, suffering from gonorrhœal cystitis, he excised, with the aid of the cystoscope, a piece of the mucous membrane of the bladder, in which he found an abundance of gonococci in the tissues of the specimen removed; some of the gonococci were seen between the epithelial cells; some had penetrated deeper into the vessels where they had produced gonorrhœal thrombosis in the capillaries and veins.

(i) *Tubercular Cystitis*.—Tubercular cystitis furnishes the best clinical representation of chronic cystitis. With few exceptions, a primary chronic cystitis is of a tubercular nature. It is important to bear this in mind in the examination of all cases of cystitis in which the initial symptoms point to a chronic inflammatory process.

Tuberculosis of the bladder is caused either by infection with the bacillus of tuberculosis through the blood, by extension of a tubercular process by continuity of surface from the kidney or the genital organs, or by the rupture of a tubercular abscess into the bladder. Vesical tuberculosis is found more frequently in males than in females, and is usually a disease of early and middle life. Tuber-

culosis of the bladder in the male is generally associated with a similar affection of the seminal vesicles and prostate. Localization of tubercle bacilli in the mucous membrane of the bladder, like that of pyogenic and saprophytic bacteria, is favored by antecedent affections of the urinary tract. Primary tuberculosis from infection through the blood is so rare that König doubts its existence. Infection takes place most frequently from the kidneys; less frequently from the prostate, seminal vesicles, and epididymis. The resistance of the mucous membrane of the bladder to tubercle bacilli is great. In many cases tuberculosis of the kidneys may exist for several years without affecting the bladder. The mucous membrane of the bladder can be irrigated with urine containing tubercle bacilli for years without becoming tubercular. Clado pointed out that tubercular granulations in the bladder do not, as is claimed by some authors, occupy the submucous tissue, but the mucous membrane itself,—that is, the subepithelial layer. He believes that this is due to the presence of a well-developed capillary net-work in the mucous membrane, which determines localization of the bacilli floating in the general circulation. Secondary infection occurs most frequently from the prostate or the kidneys, and least frequently as a result of an ascending tubercular affection of the testicles. In other cases the bladder is involved by the rupture into it of a tubercular prostatic abscess, or by the extension along the ducts to the urethra, and from there to the bladder. An ascending tuberculosis of the ejaculatory ducts in other cases precedes the bladder affection. A previous gonorrhœal cystitis not infrequently prepares the soil for tubercular infection. König observed a case in which a turpentine intoxication first produced active symptoms in a case of latent catarrhal tubercular cystitis caused by a tubercular kidney.

Although no age is exempt, tuberculosis of the bladder occurs most frequently in men between seventeen and forty years of age. Baudet records a case in a boy fifteen years old; in this case the earliest point of invasion, so far as could be determined, was the testicle, then the prostate and bladder, thence along the ureter to the kidney. The writer has seen, in a girl nine years of age, a case of primary vesical tuberculosis that extended to both kidneys and proved fatal in less than a year.

The two places in which tuberculosis of the bladder is most likely to commence are the ureteral orifices and the trigone of the

bladder. The former starting-point of the disease is the rule when the bladder becomes involved by a descending tubercular ureteritis,—that is, when the disease is secondary to renal tuberculosis; the trigone is usually the original seat of the disease in primary tuberculosis of the bladder, and in men also by the extension of the disease from the genital organs.

The tubercular disease here as elsewhere is characterized by the same chain of pathological changes,—infiltration, caseation, and ulceration. Penetration of the bladder wall frequently leads to the formation of perivesical abscess and fistula formation, a part or all of the urine escaping through the fistulous opening. The chronic inflammation and the vesical tenesmus lead to great thickening of the wall of the bladder, sacculation, and diminished capacity of the organ. The extension of the tubercular inflammation over the surface and in the direction of the different tunics of the bladder wall is hastened in case the bladder becomes infected with pus microbes, which is so often the case, and which is so frequently caused by the needless use of instruments in the fruitless search for stone in the bladder, which a beginning vesical tuberculosis often mimics so closely. The complications most frequently encountered in post-mortem examinations of patients who have died of the direct or indirect effects of tuberculosis of the bladder are tuberculosis of the lungs, kidneys, genital organs, and peritoneum, and perivesical tubercular abscesses with or without fistula formation. The disease is initiated by a frequent desire to urinate and by pain after emptying the bladder, with slight hæmaturia at longer or shorter intervals. Urination becomes more frequent as the disease advances, and after the neck of the bladder has been reached incontinence of urine becomes a conspicuous clinical symptom. The urine exhibits the same appearance and contains the same morphological constituents during the early stages of the disease as in cases of chronic catarrh of the bladder. In the beginning of the disease the urine is acid and contains pus, bladder epithelia, and a small quantity of albumen. If the kidneys are affected at the same time, the albumen is more abundant. If secondary infection with pus microbes or saprophytic bacteria has occurred, it is alkaline in reaction and often ammoniacal, and then contains also a larger amount of mucus and pus-corpuscles and disintegrated red blood-corpuscles, besides the large flat epithelial cells from the bladder. As soon as the cheesy material on

the surface of the bladder softens and disintegrates, fragments of detritus are found in the urine. Tubercle bacilli are not always present, and their detection is often very difficult. Their presence can also be determined by cultivation on artificial nutrient media and by inoculation experiments. If, in cases of suspected bladder tuberculosis, the bacillus cannot be found, the injection of a few drops of the urine sediment into the eye, a joint, the pleura, or the peritoneal cavity of a rabbit or a guinea-pig, will often succeed in reproducing the disease, and upon the results of such experiments we must then base our diagnosis. The positive results of such experiments and the detection of bacilli in the urine do not enable us always to locate the disease anatomically; in other words, we must ascertain further whether the disease involves the kidney, the bladder, or the lowest portion of the urinary tract. Nitze's cystoscope is a useful diagnostic instrument in the hands of experts. Finally, it may be stated that in all chronic inflammatory affections of the urinary organs it is necessary to make careful and often repeated examinations, both of the general and local symptoms, for the purpose of locating the disease, as well as to determine its nature, which often can be done in a satisfactory manner only by making a microscopical and bacteriological examination of the urine. If this should still leave the diagnosis doubtful, a resort to inoculation experiments upon animals susceptible to tuberculosis becomes necessary as a decisive diagnostic test.

## A CASE OF APPENDICITIS IN WHICH THE APPENDIX BECAME PERMANENTLY SOLDERED TO THE BLADDER LIKE A THIRD URETER, PRODUCING A URINARY FÆCAL FISTULA.

PAPER READ BEFORE THE AMERICAN SURGICAL ASSOCIATION, APRIL, 1898.

BY W. W. KEEN, M.D.,

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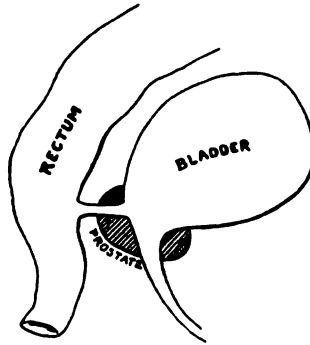
GENTLEMEN,—The following very unusual and puzzling case, in which the appendix became adherent to the bladder precisely like a third ureter and delivered fæces constantly into the bladder, is of more than ordinary interest. The difficulty in making a diagnosis, not only owing to the rarity of the case but the obscurity of the symptoms, will, perhaps, excuse the multiple operations, which were regretted, of course, as soon as the real facts became known.

Mr. W., aged twenty-four, was first seen with Dr. Charles A. Service, of Bala, on November 28, 1896. When he was seven years of age he had a great deal of trouble in passing his water, and when the family physician examined him, a pin was found well down in the urethra. He does not remember that he inserted it and believes that he swallowed it, but the only known fact is that it was found and removed. This is the only thing in his family or personal history of importance.

In March, 1896, before coming under Dr. Service's care, he was said to have had an abscess of the prostate, which burst spontaneously, the result of which was that a fistula was established between the bladder and the rectum. (Fig. 1.) No urine had ever escaped from the bladder into the rectum, but food had been frequently recognized in the urine, such as fragments of spinach, cranberry-skins, strawberry-seeds, etc. In addition to this, air sputtered out with the urine, and on many occasions the urine had a distinctly fecal odor, in consequence of which he had suffered from several

severe attacks of cystitis. When he used an enema or when the bowels were very loose, the discharge of fecal matter was very considerable, and when the fæces were solid much less escaped into the bladder. Some time ago one or more fragments of a calculus were said to have been passed, but there is no evidence as to whether they were fragments of a vesical or a renal calculus.

FIG. 1.



The supposed rectovesical fistula.

My first object was to find the two ends of the supposed fistula. He had already been seen by Dr. John B. Deaver, of this city, who, later, throughout the case, was in consultation with Dr. Service, Dr. W. J. Taylor, and myself. Examination by the cystoscope did not reveal any opening into the bladder, nor was any stone found in the bladder. Rectal touch revealed nothing abnormal. The bladder was next filled with milk, when, by the use of Kelly's tubes and the speculum of Martin, of Cleveland, I endeavored to find the rectal end of the supposed fistula by the escape of the milk while he was in the knee-chest position, but without success. On December 2, 1896, another equally fruitless examination was made with cochineal, and on December 5 he was etherized, and renewed examinations were made. The bladder was then injected with air, and the moment that this was done the perineum bulged to a considerable extent, like a cornet-player's cheek, which immediately subsided, however, and with its subsidence the air could be easily heard escaping, presumably into the rectum. This injection was made repeatedly in the dorsal, Trendelenburg, and knee-chest postures. While in the latter posture the rectum was filled with water, as I hoped that the point where the air escaped from the bladder into the rectum could be de-

tected by the small bubbles, but none were seen. Our conclusion was that a rectovesical fistula existed, and (in view of the bulging of the perineum) that in that part of its winding course it was probably not very far from the surface. We decided, therefore, upon an operation as follows:

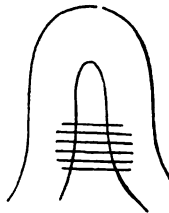
*First Operation*, February 9, 1897, Drs. Deaver, Taylor, Service, Spencer, and myself being present.—An incision was made nearly from one tuberosity of the ischium to the other, with a slight convexity forward, about an inch in front of the anus, a steel bougie being inserted into the urethra as a guide. The rectum was then separated from the bladder until finally the dissection reached to the upper end of the prostate, a depth of about ten centimetres; but at no point was the sinus discovered. During the dissection the bladder was distended with air several times, but none escaped into the operation wound. The lower border of this wound was then elevated by forceps and the wound filled with sterilized water, as we hoped to find bubbles of air in the water when air was injected into the bladder, but none were seen. Finally a perineal section was done and the finger introduced into the bladder, but nothing abnormal could be found by touch or sight. The wound was then packed with iodoform gauze and a T bandage applied. He made an excellent recovery, but the passage of gas from the urethra, heard by the nurse as well as himself, and of a considerable amount of fecal matter in the urine, which was recognized as parts of his food, showed that the fecal fistula still existed. On one occasion a small, worm-like mass passed, about one-third of an inch long and a little larger in diameter than a knitting-needle, which naturally confirmed our view of a moderately long, narrow fistula. About the first of March a sharp attack of epididymitis occurred on the right side.

After discussing all the particulars of the case, we deemed the existence of a recto-vesical fistula quite certain. It seemed to us most probable that the fistula passed from the bladder to the rectum at a point somewhat higher than ten centimetres from the perineum, but that it was so low that it would scarcely be within reasonable reach by abdominal section. On the other hand, if we opened from the perineum and opened the peritoneum, as seemed to be unavoidable, there would be the greatest danger from fecal infection. We determined, therefore, first to divert the fecal stream by a temporary

artificial anus in the left side by Maydl's operation. When this had diverted the fecal stream it was determined to reopen the perineal wound and open the peritoneum, if need be resecting the coccyx and a part of the sacrum.

*Second Operation, March 18.*—Left lateral colostomy was done. Before attempting to draw out the bowel we endeavored to see whether we could obtain any information as to the fistula through the abdominal wound, but this was fruitless. A new complication existed in doing the colostomy, since a number of strong though slender fibrous bands passed from one part of the colon to another, which made the colon assume the form of loops. (Fig. 2.) The meso-

FIG. 2.



Fibrous bands throwing the colon into loops.

colon was also so short that the colon could not be well drawn out of the abdominal cavity to any considerable extent. The bowel was then sutured after Bodine's method (*Medical News*, January 9, 1897) for three and one-half centimetres, and the loop secured in the wound by a continuous silk suture.

The result of the operation was not altogether satisfactory, as the short mesocolon retracted the spur between the upper and lower bowel so that fæces continued to pass into the lower bowel. He went to the sea-shore on March 31, 1897, for the improvement of his health. On April 8, a second sharp attack of epididymitis occurred on the left side, and the temperature rose as high as 104° F. When he returned, on April 25, he mentioned two facts which immediately arrested my attention: first, that he had repeatedly observed that he could *feel* fecal matter passing into the bladder *before any passed out at the artificial anus*, and, second, that lately, on eating some strawberries, he had *seen* strawberry-seeds in the urine *before any seeds had escaped from the artificial anus*. These two facts seem to indicate very clearly that the fistula was not between the bladder and the rectum, but between the bladder and

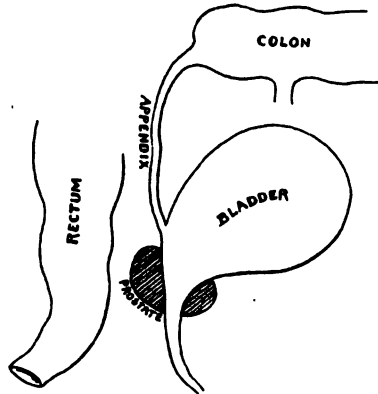


some point in the intestinal tract considerably above the site of the artificial anus in the descending colon. This great service was rendered by the artificial anus.

I decided, therefore, to open the abdomen in order to discover at what point the fistula existed.

*Third Operation, April 26, 1897.*—A median incision was made and the patient placed in an extreme Trendelenburg position. After considerable search we finally detected a very long appendix dipping down into the pelvis, the tip lying just behind the prostate, and solidly incorporated into the wall of the bladder. (Fig. 3.) That the

FIG. 3.



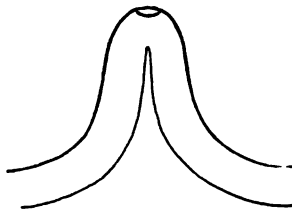
The actual condition found, the appendix being soldered to the bladder and causing a vesico-intestinal fistula.

length of the appendix and its position behind the bladder was congenital seemed to be proved by the fact that there was a normal meso-appendix dipping down into the pelvis. In order to determine whether the lumen of the appendix opened into the bladder I distended the bladder with air, but there was a difference of opinion among us as to whether the air escaped into the appendix. It only occurred to me afterwards that if we had stripped the colon downward we should have been able to empty the air it contained into the bladder. A fecal concretion lay at the middle of the appendix. Half an inch from the bladder, and with the greatest difficulty, owing to its depth, I dissected back a cuff of peritoneum from the appendix, severed the appendix from the bladder, and covered the stump of the appendix with the cuff. The meso-appendix was then divided, when, owing largely to its length, it was found to be so vascular that five

ligatures had to be used to arrest the hemorrhage. The cæcal end of the appendix was next treated in the same way. The belly was then thoroughly flushed out with salt solution and the abdominal wall closed in four layers. Before closing it we inspected the posterior wall of the bladder with the greatest care to discover whether there was any other possible point of connection between the bladder and the bowel, but found none. His temperature never rose above 100° F. For three days after the operation he suffered so severely from colic that, in spite of the normal temperature and the free opening of the bowels, I was afraid of peritonitis, but after that he recovered very nicely. I kept him in bed for three weeks, and he left the hospital May 15 entirely well. After spending the summer at the sea-shore and in Europe, he re-entered my hospital in the early part of October, 1897, when the artificial anus had contracted so that it admitted nothing larger than a No. 20 catheter. He had had recurring symptoms of intestinal obstruction on several occasions, which I was afraid was due to the spur at the site of the artificial anus, and while this had retracted enough to interfere with my purpose in making it, it had not retracted enough to leave the calibre of the bowel free. I therefore determined, he being otherwise in good health, to close the artificial anus and destroy the spur at the same time.

*Fourth Operation, October 7, 1897.*—I considered several different plans of closing the artificial anus and safely destroying the spur (Fig. 4) so as to restore the entire calibre of the bowel. I

FIG. 4.



The spur and the contracted artificial anus.

feared to divide the spur with the scissors through the small fistula lest I might open the peritoneal cavity or have dangerous hemorrhage. I also rejected Dupuytren's enterotome, and finally decided upon the following plan: An elliptical incision was made around the artificial anus, and the abdominal wall was dissected back from

the bowel about two and one-half centimetres away without opening the peritoneal cavity. The bowel was then slit up far enough to get my forefinger readily into its calibre, when the spur was drawn out through the opening and an operation identical with the Heineke-Mikulicz operation of pyloroplasty was done on the spur, the incision being made longitudinally and then converted into a transverse one and sutured with a continuous catgut suture. Several vessels bled quite freely and were ligated. I was able in this manner to determine by touch that not a vestige of the spur was left, and therefore that no obstacle to the passage of fæces remained. The artificial anus itself was now closed, first by a silk suture passed through all of the coats of the bowel and then a continuous Cushing right-angle suture. The skin was then sutured by silkworm gut. He made an uninterrupted recovery, the highest temperature being 99.8° F., and he left the hospital October 25 entirely well. The very afternoon that he left the hospital he began to have recurring pains in the abdomen, accompanied with constipation, but Dr. Service was not in the least alarmed about him till the evening of the 28th. I saw him on the morning of the 29th, and found that he had had repeated attacks of vomiting for two or three days and that the bowels had not been opened, but that gas had passed freely. The belly was not distended or tympanitic, but his general condition was bad. There was more or less pain everywhere, but not at any one special point. Possibly, had he not already had so many operations done, I might have decided to do an abdominal section at once, but I confess that in the case of a man who had just left the hospital after four operations I hesitated greatly before recommending another operation, especially in view of the fact of the free passage of flatus and that the vomitus was distinctly not fecal. On the 30th there was practically no change in his condition. Early in the morning of the 31st I had an urgent call to see him immediately, and when I got there he was in collapse. Dr. Deaver kindly saw him with Dr. Service and myself, but we concluded that the only thing to be done was to give him a saline intravenous transfusion, in the hope that his condition would improve sufficiently to allow of a celiotomy. That morning he had had a free passage from the bowels. He died at about 1.30 P.M. on the 31st.

*Necropsy Twenty-seven Hours after Death.*—The abdomen was collapsed. The moment that the abdominal cavity was opened a

few small areas of lymph, about five to ten millimetres in diameter, were noticed on the surface of the intestine in the epigastrium and the hypochondrium. The one striking fact, however, was the absolutely black color of the small intestines in the lower part of the abdomen. On examining the condition carefully, I found that seven or eight feet of the ileum had been rotated to the right in one vast volvulus. Its mesentery formed a band about six centimetres in width stretched across the ileum just before it joined the cæcum. This had evidently obstructed the blood-supply of the ileum sufficiently to cause gangrene of all the intestine involved in the volvulus, but the pressure was not sufficiently complete to prevent the passage of flatus or of fæces. This accounted for the fact that there was neither fecal vomiting nor any distention of the abdomen, and that he passed flatus constantly. From the cæcum to the bladder there stretched apparently a reproduction of the appendix which had been removed. It was impervious and had nothing to do with the volvulus which we found. The closure of the artificial anus was complete and the lumen of the bowel entirely re-established.

*Remarks.*—The original diagnosis of his first medical attendant of a prostatic abscess seems to me to have been a very reasonable one. No one could anticipate so long an appendix dipping into the pelvis and anchored by its meso-appendix immediately behind the bladder; and a terminal appendical abscess so close to the prostate might well be mistaken, I think, for a prostatic abscess. When he came to me, I confess I never once thought of the possibility of the appendix being involved, nor did it occur to Drs. Service, Deaver, or Taylor. Appendicular abscesses bursting into the bladder are not at all uncommon, but I know of no case in which the appendix has been so thoroughly united to the wall of the bladder as to form, as it were, a third ureter, delivering fæces, however, instead of urine into the bladder. Possibly such cases exist, but I have never come across them. Looking back over the case, I regret very much the perineal operation and the artificial anus. It is perfectly clear now that the first operation ought to have been an abdominal section, but in view of the symptoms, which I have detailed at considerable length, I do not think the decision reached was unwarranted.

A volvulus such as has been described I have never seen before. Unfortunately, it was not so complete as to cause entire obstruction of the bowels. If it had, I should undoubtedly have done

an immediate abdominal section, but its very incompleteness misled us as to the exact state of affairs. Whether the bands alluded to as discovered at the second operation, which were certainly very unusual in my experience, had any causative relation to the volvulus, I am unable to say, but the apparent symptoms of repeated intestinal obstruction for months are worthy of note. It is, of course, easy to say, as more than once I have said to myself, that I ought at least to have done an exploratory abdominal section, but in view of the uncertainty of the diagnosis, of the absence of fecal vomiting and other symptoms of complete intestinal obstruction, and of the fact that the poor fellow had already suffered so much in the way of operations, I think that any one of us would have hesitated to recommend a fifth operation.

## THE CHOICE OF METHOD OF AMPUTATION.

CLINICAL LECTURE DELIVERED AT THE MIDDLESEX HOSPITAL.

BY A. PEARCE GOULD, M.S., F.R.C.S.,

Surgeon to the Middlesex Hospital.

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GENTLEMEN,—It is undeniable that amputations hold a very different position to-day in the mind and practice of surgeons from that occupied by them in any former time. Amputation has been called the “opprobrium of surgery;” if this is no longer true on account of the fearful mortality attending the operation, and the sufferings and prolonged convalescence of those who escaped the many dangers of death, it remains as true as ever in the higher sense. “A great and wholly unmerited glamour has been thrown around the operation of amputation. The interest attaching to its design and execution, and to the construction of a sound and useful stump, has done much to obscure the fact that an amputation at best is a confession of failure, a refuge of the destitute, . . . a therapeutic tragedy, an irreparable disaster.” In this sense an amputation is and always will be the “opprobrium of surgery.”

But all the same, the operation still has an important place in surgical practice, and it behooves us to familiarize ourselves with its details quite as much as with those of the most beneficent and “brilliant” surgical procedure. If you take up any text-book on operative surgery you will be struck by the great number of different methods of amputating that have been employed. At whatever level or spot a limb has to be amputated the operator has a choice of methods, and in some cases a most bewilderingly large choice. I want this afternoon to give you some guide in the selection of a method, and to submit to you the grounds on which you are to select now one, now another, of these methods.

Before passing to this it will be interesting to run over the most striking facts in the history of the operation. Hippocrates—the

"Father of Medicine," who lived four hundred years before the Christian era—taught that amputation should only be performed through already dead insensitive tissues, for fear of hemorrhage. In the very early days of this era a Roman surgeon, Celsus by name, introduced the practice of amputating through living tissues just adjacent to the dead. There is some ground for the belief that he also ligatured the bleeding vessels. He was certainly familiar with the ligature applied above and below a wound of a large artery, and it is very probable that he applied the same means to arrest the bleeding from arteries in a stump. His method of amputation was what is known as the "circular" method. He made a clean circular cut through all the soft parts close above the dead tissues, then forcibly retracted them from the bone and sawed it across at a higher level. This operation is still practised; only the other day I heard of this amputation being performed through the lower third of the thigh in a case of senile gangrene of the foot. Unfortunately, the enlightened practice of Celsus was not appreciated, and we find Galen, who practised in the second century, reverting to the evil methods of Hippocrates. For the next thousand years no material progress was made, and the most barbarous processes were in vogue in place of the more merciful and wise Celsian operation. It is not until the sixteenth century that light appears in the darkness; then a great Frenchman, Paré, introduced the ligature, which has held its position in surgical practice ever since. In the next century the tourniquet was first employed, Young of Plymouth and Morel of France having the credit of independently discovering this life-saving instrument. Their tourniquet was merely a fillet fastened around the limb and tightened by twisting a rod passed beneath it,—the simplest of all the tourniquets, and one that often finds a place to-day in field surgery. It was left to Petit to devise a special instrument which effects the same purpose in a more convenient and exact way; while thirty years ago Esmarch of Kiel carried the same idea to a further point and gave us the elastic bandage and tourniquet. The flap method of amputating was first definitely practised and advocated in 1679 by an English surgeon, Mr. Lowdham, of Exeter, although there was a step towards this method when short vertical incisions were made to assist the retraction of the circularly divided soft parts from the bone. Celsus himself seems to have done this. The possibility of obtaining the requisite covering for

the bone by flaps cut in a great variety of ways has added greatly to the number of methods of amputating with which the surgeon has to be familiar. We now have flaps cut either by transfixion or dissected up, flaps consisting of skin and fascia only or of these structures and muscles also, and in some cases bone, too: flaps of equal size or of varying degrees of inequality ending in the long, single flap. And so it comes about that to-day, when we have made up our mind that an amputation is necessary, we have to choose some one method out of many in which we shall operate, and I want to give you some of the reasons which enable us to choose one or other of these various methods.

1. *The Seat of Amputation.*—Experience shows that the mortality of amputation rises in proportion as the seat of amputation approaches the trunk. The longer a stump is the greater its power and the more its movements approach the normal. These reasons lead the surgeon to amputate as low as he can and to look with the greatest regret upon any unnecessary sacrifice of limb. But he must not always be guided by this consideration, for it is sometimes necessary to amputate even far above the apparent disease. In malignant disease, for example, the surgeon must be careful not only to cut his flaps from soft parts well above the disease, but in many cases to remove the entire segment of the limb affected. Again, in cases of gangrene, where the cause of the necrosis is not strictly local, an amputation to be successful must be made high above the dead parts. For example, in senile gangrene of the foot amputation should be made through the lower third of the thigh. In these and similar cases the surgeon is influenced in his choice of site by the necessity of operating at such a level that he gives his patient a reasonable prospect of entire freedom from a return of his malady. This, of course, must be the primary consideration with the surgeon in every amputation.

There are one or two details, however, to be mentioned. Surgeons used to have a prejudice against amputating through a joint, and rightly so in the pre-antiseptic days, when suppuration followed by very slow necrosis of the articular cartilage was the general result. Now that we are able to prevent these complications we have no need to avoid disarticulations on any such ground, and experience shows that the articular ends of bones covered with joint-cartilage are especially adapted to bear pressure, and therefore make good



stumps. The more closely we study Nature's arrangements and follow her guidance the better are our stumps. We find the long bones are expanded at their joint ends where one bone rests upon another, that the hollow cylinder of dense bone becomes changed into a mass of cancellous bone with a definite and special arrangement of the bone lamellæ, and that this is closed over by a thin layer of compact bone and another layer of smooth, elastic, insensitive, non-vascular cartilage. All these changes are provisions to adapt the bone to bear pressure or to transmit weight. We find that when amputation is carried out through the cylindrical shaft of a long bone the patient is unable to bear sufficiently firm pressure upon the stump to stand upon it; but if the section is through the cancellous end of the bone the stump can bear pressure, and the nearer the section approaches the joint end the better can pressure be borne, until the very best result from this point of view is obtained when the amputation is made through the joint and all the natural arrangements for weight transmitting are preserved. We have in the knee-joint a special and beautiful example of the importance of every detail in the construction of a joint. If we amputate between the semilunar cartilages and the tibia,—leaving those cartilages upon the femoral condyles, whose rounded surfaces they render flat,—we find that the patient can afterwards rest his weight upon the end of the stump with far more security and comfort than if we remove these cartilages. To put this matter very briefly and practically, a man whose thigh has been amputated through the shaft of the femur cannot stand upon the end of his stump, but has to transmit the weight of his body from his ischial tuberosity to the artificial limb, and the length of stump is important to him as leverage only, whereas if his leg has been amputated below the semilunar cartilages he can rest his whole weight upon the end of the stump without any discomfort, and he stands and walks upon his stump.

In amputations of the hand there is another point to be borne in mind. If we amputate through the wrist-joint the patient preserves the movement of pronation and supination, a matter of great importance; but if the surgeon saws off the joint ends of the radius and ulna this movement is lost. In some cases the carpal bones can be preserved, and then the flexion and extension movements of the wrist can be communicated in some slight measure to the artificial hand.

In other cases the operator has to bear in mind the attachments of muscles in selecting the site of his amputation so as to preserve certain movements in the stump. The best example of this is at the knee. If the section is made above the adductor tubercle to which the tendon of the adductor magnus is inserted, the patient loses the power of thrusting his thigh straight forward in walking, and he swings his stump round outward and forward. The division of the femur half an inch higher or lower, just at this spot, will make all the difference between elegant and inelegant walking.

2. Having decided at what level to amputate, the surgeon must next make up his mind how he is to *obtain a sufficient covering for the stump*. The ideal to aim at is to have the end of the bone covered over by loose soft parts, with the line of scar neither adherent to the bone, over its end, nor subject to pressure, and to form the covering from well-nourished parts. If the stump is one on which the patient is to bear weight, the skin should be taken from some part used to, and specially fitted to, withstand pressure. This ideal is best obtained by dissecting up carefully designed flaps which can be brought together with accurate fit, as in Syme's amputation at the ankle or Teale's amputation. But there are two or three conditions which in certain cases must influence the surgeon. Thus, either from injury or disease the soft parts may be so destroyed that it is impossible to dissect up such flaps without amputating at an undesirably high level. We must not unnecessarily maim a patient in order to secure an ideal stump, but, of course, a long stump, if "sugar-loaf" or tight, or with adherent and often ulcerating cicatrix, is also quite useless. We have to weigh carefully the *pros* and *cons*, and with ingenuity it is often possible both to preserve a long and strong stump and to get for it an ample and efficient covering. Very rarely in civil practice, but often in military surgery, speed is an element to be taken into account. It takes much longer to dissect up accurately designed flaps than to cut two flaps by transfixion, or to execute the old Celsian operation, and where there is no anæsthetic, or where it is necessary to get the operation over as speedily as possible, one or other of these methods should be used. In the days of our grandfathers surgeons amputated as men now race, with a time-keeper looking on to record the exact number of seconds taken, and some of them attained to remarkable speed in the transfixion flap operation. A man whose thigh was amputated in one of the

London hospitals without an anæsthetic told me that from the moment of his entering the theatre to the time of leaving it with his stump dressed was only eight minutes! To-day we have none of this extreme rapidity in operating; our aim is rather to operate with the extreme of accuracy.

The next point to determine is the thickness of the covering of soft parts: should it consist of skin and fascia only, or of these and the muscles also? If we include muscle in our flaps, it makes a thicker covering for the bone at once, but the muscular tissue wastes and ultimately disappears, so that it is of no permanent value. But as the blood-vessels of a limb run between muscles, whenever we cut a muscular covering we are sure to have it well supplied with blood, whereas if we strip up long flaps of skin and fascia only the blood-supply to them is small, and unless great care is taken in the operation more or less sloughing may occur. Large muscular flaps are not good; they are troublesome to adjust accurately, and, if any swelling subsequently occurs, the muscle bulges between the sutures. In any case the absorption of the muscular tissue takes time, and it is long before the stump arrives at its final condition and is ready for the fitting of an artificial limb. Flaps of skin and fascia are easily adjusted, and as there are no later changes which alter their shape or bulk, the stumps are earlier ready for the instrument-maker. The skin of the back of the hand is thin, ill-nourished, and makes bad flaps, the skin of the palm is not only thicker, but is well supplied with blood-vessels, and it is easy to preserve the vessels in the flap; it makes good flaps. The skin of the heel, too, is well supplied with blood, and makes a good covering to a stump. The skin of the front of the knee also is well adapted for flaps, being well nourished as well as thick and adapted for pressure. My own preference is for flaps of skin and fascia with circular division of the muscles, and in many cases I saw the bone at a higher level than I divide the muscles. There is room for considerable variation in this matter, for skin and fascia may be dissected up for a short distance and then the line of section carried gradually, obliquely, through the muscles to the bone. The circular method in two or three sweeps of the knife accomplishes just this result, but with flaps—even short ones—it is easier to get a perfect fit of the parts, and by adaptation of the length of the flaps we can secure that the line of cicatrix is not just over the bone. In these particulars the circular method

is inferior. But it has one merit in which it stands alone, it takes its covering equally from every part of the circumference of the limb, and therefore enables us to amputate lower down a limb—nearer to an injury or disease—than any flap method: its antithesis in this particular is the single-flap method, such as that of Carden at the knee or Farabœuf at the upper part of the leg.

The last point to which I will refer to-day is the advantage of lessening the number and size of blood-vessels and muscles divided in a stump. It is for this reason that Spence's method of amputation is to be preferred at the shoulder and Furneaux Jordan's at the hip-joint. Not only is the healing of the wound promoted by lessening its area, but the shock is diminished, which, at the hip-joint, is an important matter. In cases of sarcoma of the shaft of the femur it is often impossible to employ the Jordan method, but the principle can be carried out in another way if we reflect the skin and fascia by means of an oval incision and then cut the muscles quite short close to their attachment to the pelvis. Thus performed, the operation is attended with less shock than if large muscular flaps are cut by transfixion, and a very satisfactory stump is obtained. This method may be called the "modified oval" method, and is similar to the "modified circular" method with which you are more familiar. Of the many smaller details of amputations I must speak to you as opportunity arises in the wards.

## TREPHINING FOR TRAUMATIC EPILEPSY.

CLINICAL LECTURE DELIVERED AT THE KENTUCKY SCHOOL OF MEDICINE HOSPITAL.

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GENTLEMEN,—The history of the case I will give you briefly as the patient is being anæsthetized. A man, aged forty-one years, received a blow on his head ten or twelve years ago; he was found unconscious at his barn: it is presumed that he was struck on the head by a negro who was at the barn and who was in his employ at that time. The man was taken to his house in an unconscious condition and remained so for several hours, his brother who brings him here not being able to give us any definite history on this point. He regained consciousness, however, after the lapse of several hours, and suffered from headache, fever, and other symptoms which kept him in bed for about a week, his brother thinks. There was no wound of the scalp so far as he knows, but there is a visible scar at the present time to indicate the existence of one.

The man had the first seizure of an epileptic nature about five years ago while driving with his wife in a buggy; he fell, and was entirely unconscious, having had an ordinary epileptic seizure. Since then these attacks have grown far more frequent. At first there were months between the attacks, but at the present time he is having one, two, three, or more attacks during each week. Nearly all of his recent attacks have been at night, he rarely having one during the day, and this I regard as rather unfavorable, for a case of nocturnal epilepsy is always a little more unyielding to treatment than the diurnal variety. Unfortunately, his wife, who is the one who knows most about his condition, is not here, and his brother is not able to tell us how the paroxysms are initiated; he cannot say whether they begin in the arm or leg, whether the seizures are local or general; or, if general, whether they are preceded by local spasm of one arm or one

leg. The paroxysms occurring as they almost invariably do, however, at night, perhaps even if his wife were here we could not get any very definite history from her. The man is totally oblivious of his condition during these attacks; he only knows that he has had one the night before by feeling very sore the next day. The case is unquestionably one of traumatic epilepsy; only this is certain.

You may ask why do I say that he has traumatic epilepsy, why might it not be a case of ordinary idiopathic epilepsy in a man who has previously received a blow on his head, for, as you know, very few of us as boys have not been struck on the head in a fight or in play, and we might well suppose that this man could have had epilepsy independent of the traumatism. There is one feature that guides us very positively, and that is a tender painful cicatrix. As soon as we make pressure at this point the patient evinces pain, showing that it is very sensitive, and he says that he constantly suffers with pain at the site of the depressed bone and on the entire left side of the head. Pressure upon this painful cicatrix does not, as in some instances, provoke a spasm. Therefore I take it that, having marked local tenderness always existing at the site of the wound, being sometimes very severe and being most positively demonstrated by pressure, and at the same time having evidence of depressed bone, there can be little doubt as to the diagnosis in this case.

The next question that comes up is, What was the diagnosis primarily? This feature concerns us more or less, and I therefore wish to digress for a moment. From what did this man suffer at the time of the injury? I believe that he had concussion of the brain; I am not prepared to say that he had compression, because there is no history of paralysis either of short or long duration, and in the absence of that history I take it that the man had rather a severe and unusual attack of concussion of the brain. What should have been the treatment at that time? If the man had a depressed fracture, and it was marked, as it must have been, even more marked than at the present time, because there is a certain amount of resiliency of the skull, and it will elevate itself (especially in young subjects) in a way after a time; if there was a depression even so marked as now appears, followed by complete loss of consciousness, as there was in this case, even though consciousness returned in the course of an hour or so, I believe, in the light of modern experience, that this man should unquestionably have been trephined

at that time. If he had been, he might have been saved from epilepsy, which he has and may have the rest of his life, perhaps.

The treatment of depressed fractures of the skull has undergone a most decided change, therefore I wish to explain to some of the older gentlemen in the class who may have read the literature of the subject which teaches just exactly the reverse to what I have said should have been done in this case. There are a few cardinal rules or principles in brain surgery upon which all of us are agreed. There are others which are mooted questions; there are others still upon which we are diametrically opposed to one another. In the first place, we are all agreed that in punctured fractures of the skull trephining is the only thing to be considered. Anything less than trephining in punctured fractures of the skull is out of the question; it would simply be malpractice.

Then in compound depressed fractures of the skull with symptoms of compression, all agree that immediate trephining should be done. In compound depressed fractures of the skull without symptoms of compression, the majority of modern operators would wait until symptoms arise, and then if compression is evident trephine the skull. This latter practice is very objectionable, highly so on account of the fact that the mortality which follows operations done in the inflammatory stage is very great, being ten times as great as when done primarily. Therefore, I believe, one is not justified in waiting for symptoms in case of a compound depressed fracture of the skull. We are all pretty nearly together so far. Then we come to simple depressed fractures of the skull with symptoms of compression. Here there is some doubt, but the majority of modern operators, I take it, would say operate, even though there is no wound of the scalp. The old objection that you convert a simple into a compound fracture does not amount to anything if you do the work aseptically.

This patient did not have a punctured or a compound depressed fracture, with or without symptoms, or a simple depressed fracture. He may have had some symptoms of compression, in fact, I rather incline to the view that it was a mixed case, between compression and concussion, but in the absence of a positive history of paralysis, etc., and his regaining consciousness as soon as he did, we must rather call the case one of concussion and not compression. Here we have to deal with a simple depressed fracture of the skull, without, we will say, symptoms of compression. What should be the treat-

ment? Here you find the profession divided; one side says that you cannot have depressed bone to any extent without necessarily damaging the brain or its membranes, therefore you will not wait for symptoms, but anticipate them, because, even if the man escaped the primary dangers of compression, concussion, and encephalitis which follow in the wake of it, he is liable to have a tumor, an abscess, or is liable to become an epileptic. Now we are coming to the case in point, for that reason they say trephine even for simple depressed fractures of the skull without symptoms of compression. I unhesitatingly place myself among this class, and think that is the way these injuries should be treated. I am also pleased to state that my preceptor and cousin, Dr. W. B. Rodman, of Frankfort, Ky., was, perhaps, one of the first men in this country to advocate such radical treatment in simple depressed fractures of the skull. He published an article in the summer of 1877 in the *American Journal of the Medical Sciences* advocating this practice, and he was naturally assailed by many surgeons in the country, the elder Gross particularly being quite caustic in his criticism, but that practice to-day is the practice of modern surgeons, not only in this but other countries,—trephining for depressed fractures of the skull without symptoms of compression.

Suppose you have another case, a linear fracture of the skull without any depression; none of us will trephine in such a case unless the fracture is compound. But if there is much depression of bone, there must be necessarily injury to the brain and the membranes beneath, and while the fracture of the bone may not amount to much the accompanying lesion of the soft parts may be very serious. Another thing you should remember is that the skull is sufficient to protect the brain from ordinary violence, encasing it in a bony envelope, and it is only in cases of unusual or extraordinary violence that it fails to give adequate protection. You should also remember that this protection, which is most desirable and necessary, is only gained at the expense of lack of drainage, when that may become necessary to save life. So there are disadvantages as well as advantages in having the brain so thoroughly encased. Therefore it is to facilitate drainage that we remove a portion of the bone and tap an abscess, cyst, or remove blood-clots, etc.

What are we going to find in the case before us? I do not know. I suppose we will find a depression in the inner table, just as we have found in the outer table, but even if we find a depression of



the inner table, we will not be satisfied if there is more opacity of the dura mater than there should be and especially if there is absence of pulsation of the brain, or if there is lividity or yellowness of the dura mater; any or all of which symptoms would indicate a lesion beneath the dura. We shall not be satisfied if any of these conditions are present until we have incised the dura mater,—which incision should be made in the shape of a semicircle, keeping at least one-quarter of an inch away from the opening made with the trephine,—and inspected the brain and meninges beneath the dura. What shall we be likely to find in this case? The most probable thing would be a cyst. It is very common after a hemorrhage, and we might have had a subdural hemorrhage here. A cyst often follows where a clot is breaking down and becoming absorbed. So we may have pressure from a cyst in this case. I take it there is no abscess. And I wish to say that abscess of the brain is by far more difficult to diagnosticate than deep-seated suppuration anywhere else in the body. For some reason there is entire absence of chills, sweats, pain, and all the ordinary symptoms which come with deep-seated suppuration. The reason I make this statement is that you may operate upon a head case without symptoms, and most unexpectedly come in contact with pus. If we knew that this man had paroxysms initiated by or beginning in the right leg, and the lesion is near the situation to cause such spasms, then I should not hesitate to excise the leg centre; but we do not know that, and not knowing it, I am unwilling to excise the leg centre. We will subject him to just as little risk as possible.

We shall make the trephine opening with a three-quarter inch trephine, then enlarge the opening with rongeur forceps, or with mallet and chisel, as we deem advisable. The head has been thoroughly sterilized, first by a bath in bichloride solution, after that a poultice of soap was left in contact with the shaven scalp for twenty-four hours, and the soap in this case proved a little irritating and blistered the scalp, which it will do sometimes. We will now re-sterilize the field of operation with soap and water, and after doing that Dr. Laws will use ether, so as to get rid of any epithelial scales which may be present, and when this has been done we shall be prepared for a complete operation. We cannot tell just what it will be necessary to do, and my judgment is that in cases of this kind one should always be prepared for any emergency. We might content ourselves, as we did in one case which you will remember last

session, by only excising a cicatrix in the soft parts. Or we may go ahead and take out the bone with entire satisfaction, as we did in two other cases; or again it may be necessary after opening the dura and locating the arm centre by the stimulation of the battery, as we did in one case, to excise the cerebral cortex containing the centre itself.

Chloroform is the better anæsthetic for brain operations. It is less of a heart stimulant, and on that account less blood is carried to the brain. The flap should be made with the base downward, so as to interfere as little with the blood-supply as possible. The flap should be made horseshoe or semicircular in shape. The old method—and why it should have been popular I cannot understand—of making a crucial incision is out of the question and no longer practised. The horseshoe flap should be made with its base downward. Make one cut down to the skull including the pericranium. Hemorrhage can be controlled without difficulty by forceps; but another very good way, and I am going to use it to-day, is to throw a rubber tube around the scalp. It is a method used by Keen, of Philadelphia, and while less hemorrhage is not an especial item, still this man is a little thin and anæmic looking, and we will make the operation as nearly bloodless as possible.

Having made my incision, you will observe that the depression in the skull is very marked. The scalp also shows a distinct scar, which may or may not have embraced all the scalp tissues. The thing to do in these operations is just as much as is necessary and no more; timidity is just as objectionable as being too rash and bold, for the timid operator will sometimes fail to achieve success where a bolder plan would have achieved it. We have made a horseshoe incision, including the pericranium, and turned the flap downward. We have freed the bone from its pericranium, and now proceed to apply the trephine. We should not make the trephine opening right over the depression; we go to the edge of it. When you have once made a groove with your trephine, get the bit out of the way else you do harm rather than good. You have done all the good you can with the bit, and you may do harm if you allow it to remain in the instrument. The skull at this point in the white man is on an average of one-fifth of an inch, sometimes half an inch, sometimes less than one-eighth or one-tenth of an inch in thickness, varying very much. As long as the bone-dust from the trephine is white, you know that you are in the bony part of the skull; as soon as it be-

comes red you are in the diploe; then certainly, if not sooner, the bit should be withdrawn. If you proceed ever so carefully with the trephine, you will sometimes cut more on one side than the other; and if the instrument is held evenly one side will occasionally be cut through before the other. This is due not to any fault of the surgeon, but to the inequalities of the skull itself, as it varies in thickness and is irregular as we know. In these cases it is better to operate too slowly than too rapidly. I have now removed the button of bone, and it will not be replaced. We could at once immerse it in a one to two thousand bichloride solution and successfully implant it, but for manifest reasons we will not do so in this case. The opening in the skull will now be enlarged with mallet and chisel by preference. You will observe that the chisel cuts very readily. The blows should be delivered obliquely in using the mallet and chisel, so as to cause less shock. We find that the brain does not pulsate; it also bulges into the trephine opening, and while I am not ready to say there is a cyst beneath the dura, I begin to strongly suspect it. After trimming the bone down very thin with the chisel, with rongeur forceps we bite it off and so enlarge the opening without any trouble. It is necessary to proceed very cautiously. We find there is considerable depressed bone, and it requires infinite patience to remove it without injuring the dura, which is adherent at some places. The entire dura mater that can be inspected is more congested than is usual. A small vessel in the diploe is bleeding, which we cannot tie, nor catch with forceps, so we will stop the hemorrhage by stuffing a little piece of catgut into the opening. The dura does not pulsate; it feels tense, and bulges into the trephine opening. There is marked congestion everywhere, one point of the dura is markedly adherent, and while we might stop the epileptic convulsions entirely by concluding the operation where we are now, there should be pulsation of the brain, which I fail to get, and I believe I will hardly do my duty to this man unless I open the dura. With very fine catgut I will ligate a small vessel which I see in the dura before incising it. As already indicated, the dura should be cut at least one-quarter of an inch from the margin of the bone, otherwise we would have almost insuperable difficulties in attempting to suture it. We have opened the dura, and find there is a cyst as large as a small walnut. Those of you who are near enough to see will observe that the brain itself looks milky, opaque, or yellowish. I am now going to open the cyst, and for this purpose we will use a small

hypodermic needle which has been thoroughly sterilized. The fluid has been withdrawn and the demonstration is perfect. We should not have done this man any good if we had stopped without going into the dura. We have removed all the depressed bone and emptied the cyst, which leaves a depression in the brain at that point. The dura is very much thickened. The brain no longer bulges into the opening, and we have no trouble in suturing the dura. If we had done as I did in the first case of this kind I ever operated upon, cut too close to the bone, we would have been embarrassed, as I was then, in applying our sutures; in that case I finally sutured the dura, but it was at the expense of considerable time and trouble. The dura should be sutured with ordinary catgut, never use hardened or over-prepared gut, as we want a suture that will be absorbed and pass away quickly. Chromicized gut has no place here. The dura can only be sutured with a very strongly curved needle, and in this case we apply interrupted sutures. The cyst is drained by means of a small piece of rubber tubing sterilized by boiling in bicarbonate of sodium solution.

Now you see, gentlemen, that this case has turned out so far very well. We started out by being perfectly honest, and stating that we did not know just what we were going to do; that we were simply going to meet the indications as we progressed. We rather hoped it would prove unnecessary to enter the dura, and yet we told you that we would do so if we thought it best. Cutting down upon that membrane, it was plainly evident to me that less than that would do the man no good, therefore we did not hesitate to open the dura. The operation thus far has necessarily been done slowly, as it is a very serious matter to open the brain as we have done; the balance of the operation will be quickly completed, and the wound will then be closed. We have used warm water in irrigating the field of operation so as to avoid shock as much as possible. Some of you will remember that last year we operated upon a patient after about the same fashion, except that we went a step further and removed the leg- and arm-centres; he suffered from severe shock (making a good recovery, however); in this case we will avoid that if we can. These operations are oftentimes followed by so much shock that some surgeons think it is best to do them in two sittings,—that is, remove the bone, then put in preliminary sutures, and at the end of seventy-two hours reopen the wound, incise the dura, and complete the operation. I do not think this is the best plan. I can well understand, however,

in some cases where it would be perhaps best, especially in resecting the Gasserian ganglion, which is done in two sittings by many operators. In the case before us we are perfectly satisfied we have done our duty, whatever the result is. The chances are ten to one, however, that he will make a good recovery. He leaves the operating table with a pulse of 80, and is in splendid condition. Should he not recover, we have the satisfaction of knowing that we gave him the only chance in the world that could possibly bring about an amelioration in his condition. That he was getting steadily worse was noticed both by himself and his friends. At the same time, knowing what the final result of epilepsy is,—imbecility in the vast majority of cases,—any measure which promises betterment, if not cure, is justifiable. We have a reasonable chance for a cure in this case. A similar case operated upon seven months ago has not had an attack since. Before the operation he had as many as fifty a day. Even if we have not effected a perfect cure in this case, I have not the slightest doubt that we will have benefited the man. I am sure his attacks will not be so frequent nor severe, so if we accomplish that in a terrible disease like epilepsy we have done something. There is no chance of his being made worse; if he survives at all he will be better, and possibly be entirely cured. We shall hope for the best result as a matter of course, and yet I tell you frankly that surgeons of the greatest experience in this class of work are very guarded in making a prognosis. I am not going to tell the brother that this patient will be positively cured; we cannot make any such promises, because the case to be entirely cured must pass the two or three years' limit, just as in an operation for malignant disease. For instance, you take out a cancer of the breast to-day, the patient recovers from the operation without any trouble, and goes home at the end of two or three weeks. We cannot say to the patient that it will not return; the best we can say is that, if the growth does not recur within a year, the patient is comparatively safe; if she goes two years without recurrence, then you can count reasonably on her being safe; if there is no recurrence in three years, then you are almost certain that the growth will not recur.

The question will naturally come up in your mind as to what can we do in a case of what is called medical epilepsy,—that is, the non-traumatic variety. Nothing at all, unless it is purely an epilepsy of the Jacksonian or focal type. Some of you may not know what we mean by Jacksonian epilepsy; we mean by that a case where the



**FIG. 1.—Scar resulting from a successful case of trephining for Jacksonian epilepsy.**



paroxysm is always limited to a certain set of muscles; that it does not extend to other muscles, but the spasm is limited to the arm or leg, and does not become general. These cases can be operated upon, some few are cured, others are benefited, a still larger number are not improved at all. Then, again, you have focal epilepsy, where the convulsion becomes general, the whole body becoming involved, but the spasm starts always in a certain set of muscles. It may begin in one arm or leg, afterwards extending over the entire body. You will remember the boy we operated upon last spring, removing the arm-centre, as in his case the spasm always commenced in the right arm. That was purely a case of focal epilepsy. We are perfectly justified in operating upon such cases, but, of course, we should be very guarded in our prognosis as to the final result.

The reason why, in my judgment, there are so many cases of traumatic epilepsy that are not benefited by operative measures, is because of the timidity of the surgeon; they do not open the dura, and therefore do an incomplete operation, leaving a cyst or blood-clot or something else beneath or in the dura itself. In other words, the cause of the epilepsy is not removed; it is an incomplete operation, and they can only expect partial results, if any at all. I believe that if the rule should become general, which I confidently expect it will in time, to open the membranes in all cases, we shall get excellent results in trephining for traumatic epilepsy (Fig. 1). It is always best to do as we did in this case, make a good, large opening in the skull,—you cannot work through a little half inch opening. You want plenty of room, so that you can control the hemorrhage, and inspect the brain carefully to see what the trouble is, then set about to right it as best you can.

A few words as to cerebral localization. In this case the trouble is located well back of the fissure of Rolando, the most important of all the fissures of the brain. Dr. Dunn was kind enough to prepare the chart, which I show you, on which he has outlined the fissure of Rolando and located the principal brain centres in this region. He has marked for us the shoulder-centre, the arm- and leg-centres, then farther down and in front we get what is known as the Broca convolution or the centre of speech. The lesion in the case just operated upon is just behind and below the arm- and leg-centres, possibly encroaching upon the latter slightly. It would not seem so, however, as the battery applied at the site of the cyst gives no response. It is probably in what is called a latent zone of the



brain. Keen's double electrode is indispensable in these operations.

The fissure of Rolando is easily mapped out; all you have to do is to measure from the glabella to the external occipital protuberance, which I did in this case, and found it was thirteen inches; then you take one-half of that which would be six and one-half inches, then going just one-half inch behind this point, you would find the beginning of the fissure of Rolando. Its course is then indicated by a line three and three-eighths inches long, at an angle of 67 degrees downward and forward. If the battery is to be used, and I always prefer to do so, as in this case, the current should not be too strong, just sufficient to bring the thenar muscles into play. You saw me do this before applying the double electrode to the brain. Antiseptics prevent a satisfactory response from the current. I believe their use unnecessary anyway, perhaps harmful.

[NOTE.—The patient left the hospital fifteen days after the operation without having had a convulsion. The wound healed by primary union. A year subsequent to the operation he is entirely well and has only had one convulsion, and that a short time after leaving here. He has gained flesh, is in fine general health, and in every way a different man. His brother, who is now under my care, tells me that the change in his condition has been remarked upon by every one.—W. L. R.]

## **SYPHILITIC STRICTURE OF THE RECTUM.**

CLINICAL LECTURE DELIVERED AT ST. GEORGE'S HOSPITAL.

**BY T. PICKERING PICK, F.R.C.S.,**

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GENTLEMEN,—My attention has been much directed recently to the subject of so-called syphilitic stricture of the rectum, on account of my having had several cases under my care, and a study of these cases has compelled me to come to the conclusion that the explanation of the causation of this affection, as given in our ordinary text-books, is by no means satisfactory. The fact that I have at the present time a patient suffering from this affection in Drummond Ward, whom you can examine for yourselves, gives me an opportunity of bringing the subject under your notice and of discussing with you the grounds I have for thinking that the usual explanation of the etiology of the disease requires further elucidation, and also of briefly alluding to the treatment of this affection.

If we refer to what has been written on the subject we shall find that almost invariably it is said to be due to a gummatous infiltration of the submucous tissue of the rectum. Mr. Alfred Cooper in the last edition of his work on syphilis, which has recently been published, says, "Gummatous deposits and infiltration of the submucous tissue are the most common causes of syphilitic ulceration and stricture of the rectum." Erichsen says it is "always a tertiary affection, and is seldom associated with any affection of the vagina." McCarthy, in Heath's Dictionary, says, "The submucous tissue may be uniformly infiltrated so as to produce rigidity and stenosis of the bowel, or gummata of large size may cause projections into the lumen of the tube, or numerous minute gummata may form and gradually extend for a considerable distance up the rectum." Jonathan Hutchinson, Jr., in Treves's "Surgery," says, "We believe that tertiary syphilis is the most important cause of non-malignant strict-

ure of the rectum, and the lesions which may produce it are well recognized." I am bound to say that I have stated the same thing myself. In the last edition of Holmes's "Principles and Practice of Surgery," which I had the honor of editing, I am responsible for the following passage: "It is essentially a tertiary affection, and consists in a fibroid induration of the submucous tissue, with ulceration of the mucous membrane." This passage was written some years ago and before my attention had been particularly directed to this subject. In any future writing on this point, I should feel compelled to very considerably modify this statement.

Many other authors might be quoted, but I think I have brought forward sufficient evidence to prove that there is a very general consensus of opinion in favor of syphilitic stricture of the rectum being a tertiary affection and due to a gummatous infiltration of the submucous tissue. It is to this opinion that I demur, and my principal grounds for doing so are as follows:

The disease almost invariably attacks females, below the age of thirty-five, and in these two facts we have powerful arguments against its being a tertiary affection. Let us take the question of sex first. As I have said, it almost invariably attacks *women*, and I think I might have left out the qualifying adverb and have said it invariably attacks women; at all events, I cannot remember any instance of the disease as it is commonly seen in women, consisting of ulceration, followed by cicatrization and stricture, as having occurred in the male sex under my own observation. Now, if this is a tertiary affection, due to constitutional syphilis, to which men are certainly as liable as women, it is difficult to account for this fact. Why should not men suffer as often from the disease as women?

Then, again, as regards the age. This disease occurs for the most part in young women, often under the age of thirty, but gummata are among the later manifestations of syphilis. Carry your minds back over the cases of gummata that you have seen and you will remember that they occurred principally in patients somewhat advanced in life. We find gummata in the tongue and in the muscles of patients over fifty, rarely in patients under thirty. Why, then, should they appear in the rectum at this early age?

Again, there are other arguments against the gummatous nature of this disease. It is astonishing in how many of them there is an entire absence of any history of previous syphilitic symptoms or of

any manifestations of syphilitic lesion in other parts of the body. Since my attention has been particularly drawn to this subject, I have investigated the history of these cases with the most scrupulous care, and in most of them have failed to obtain any history of any secondary lesions, even when the patient was evidently desirous of giving the fullest information and showed no disposition to conceal anything from one. Then, again, I have most carefully examined the body to find evidence of any trace of former syphilitic lesions without success, and one knows how common it is in tertiary affections to find the remains of some former syphilitic trouble, which often assists us in forming our diagnosis. Again, the general appearance of the patient is against the view that this is a tertiary affection. It will be conceded by all, I think, that most patients who are the victims of tertiary syphilis present evidences of being broken down in health; they present an anæmic, emaciated appearance, which does not tally with our ideas of robust health. It is far otherwise with those young women who are suffering from this so-called syphilitic disease of the rectum. In the early stages, before their health has been broken down by the profuse discharge which occurs in these cases, they are often seen to be robust, red-faced, and plump, apparently in the condition of perfect health. Later on, it is true, they become worn out and emaciated, even to a very remarkable degree; but this is the result of the local disease and not of any constitutional cachexia. There is another peculiarity in these cases which to my mind is a further indication that they are not, as a rule, cases of tertiary syphilis, and that is the result of treatment. We all know how efficient iodide of potassium is in cases of tertiary syphilis of all kinds. Either alone or combined with mercury in almost all cases it does some good and in most it effects a cure, so that we constantly see large gummata rapidly disappear under its use. So much is this so, that we are constantly in the habit of giving iodide of potassium for diagnostic purposes. Given, let us say, a case of doubtful ulcer of the tongue, doubtful, I mean, as to whether it is epitheliomatous or a gumma, what is our usual course in order to clear up the diagnosis? We give iodide of potassium for a week or ten days, under the distinct belief that if it is of a syphilitic nature we shall find marked improvement under its use; whereas if it is malignant there will be no improvement, but rather an increase of the disease. Now, what is the result of the administration of iodide of

potassium in these cases of so-called tertiary syphilitic disease of the rectum? In my hands it has been most unsatisfactory. In many it has seemed to do no good at all. I have had a woman, aged about thirty, under my care for the last three or four years as an out-patient, who has been taking either iodide of potassium or iodide of sodium almost continuously during the whole of that time and in whom the ravages of the disease are still going on. At one time for some considerable period she took mercury in combination with her iodide, and during most of the time she has applied mercury locally to the part in the form of the blue ointment or oleate of mercury introduced on a bougie. Now, if this was a tertiary syphilitic affection, one would have expected it to be well under this treatment long ere this. But it is not so, and I could quote many similar cases which have occurred under my own observation, all pointing in the same direction,—that iodide of potassium does not effect any beneficial action in these cases.

Finally, if the condition is cured, the patient entirely recovers and becomes plump and strong and shows no further indications of developing tertiary affections, which would probably not be the case if the disease was of constitutional origin. Some of you may remember a woman who occasionally comes to see me in the Surgery with an artificial anus in the left groin. She is a typically healthy-looking woman, stout, robust, and certainly not looking like a person who was the subject of tertiary syphilis. This patient came under my care in the first instance about eight years ago, suffering from this so-called syphilitic disease of the rectum, which had first appeared shortly after her marriage. She was placed under the ordinary routine treatment, but without any benefit; in fact, she steadily got worse, fistulæ formed and burrowed in every direction; she had profuse discharge, and became hectic and emaciated to a degree, and was evidently sinking from the effects of the excessive discharge, when about four years ago I suggested to her that I should perform inguinal colotomy, in order to divert the passage of the fæces from over the ulcerated surface. The operation was done, with the happiest results; the ulceration rapidly healed, and ever since she has enjoyed perfect health and has shown no indication whatever of any syphilitic disease. She passes all the motions through the artificial anus, and simply washes through the lower part of the bowel once a week with some solution of boroglyceride to get rid of a little

mucus which collects in the gut. The ulceration of the rectum is all healed, but the canal is so narrowed that it will scarcely admit a No. 12 catheter.

I think I have brought forward sufficient evidence to make us pause before we receive the usually accepted opinion that this disease is a tertiary syphilitic affection. But if it is not this, what, then, is it?

In some of the text-books from which I have already quoted there is another view put forward about these cases, but it is only put forward to be placed on one side as improbable. This view was, I believe, first suggested by Dr. Gosselin, of Paris, and it is that the disease is not syphilis at all, but is a chancrous ulceration extending from the vagina, either by the discharge running down to the anus, or through menstruation, or, it may be, by direct contact from the male organ. The disease is, I should mention, almost exclusively met with among the poorer classes and in hospital practice, and it is suggested that these women are liable to get various forms of disease about their genitals, and it is said that when they get soft sores upon the labia or in the vagina the discharge from these sores is apt to inoculate the anus, spread up the rectum, and so give rise to this condition. In connection with this view Erichsen says, "There is no evidence to support it," and Christopher Heath says that "This has always seemed to me a very unsatisfactory explanation." For my own part, it seems to me to be a more satisfactory explanation than the one which assumes that it is a gummatous infiltration. I have endeavored to show that it is not the latter, and clearly it must be due to some cause, and this seems to me a probable explanation, viewed especially in the light of a careful investigation of cases. Take, for instance, the case of the woman who is at present under my care in Drummond Ward. She tells me that she had always enjoyed good health until her marriage five years ago; that shortly after this she was attacked with sores about the vulva, and that subsequently the disease commenced in the rectum. To use her own expression, which was entirely voluntary and not in any way suggested by myself, "The sores on her privates extended to her back passage." She is a woman of thirty-five years of age, and has absolutely no indications, nor can I obtain any evidence, of syphilis. She has never been pregnant. And the history of this woman is the history that you will often obtain in these cases: that first of all

there are sores on the vulva, often appearing after marriage, and that subsequently the patient begins to suffer from difficulty and pain in defecation and the passage of bloody discharge during the action of the bowels. If we adopt this view we at once explain why the disease occurs in women, and especially among the poorer classes, who are more apt to get sores about the genitals, and who probably are not so careful when they do so as to cleanliness and treatment.

The disease, then, I believe to be a chancrous ulceration extending from the vagina, and its continuance and reluctance to heal is due to the diseased surface being constantly irritated by the passage of the *fæces* over it. To this latter point I will allude more particularly when I come to speak of the treatment. But before I do so I am bound to mention another view as to the pathology of this affection which has been put forward by Fournier. He believes that the disease is due to the infiltration of the wall of the gut with a new formation, and to it he gives the name of anorectal syphiloma. He states that this new material undergoes fibroid changes and produces contraction of the calibre of the canal, and that, as a rule, there is neither ulceration nor cicatrization in these cases. My answer to this is that such a case is very different from the ones which we have now under consideration, where there is undoubtedly ulceration and profuse discharge. I have seen cases similar to those described by Fournier, but they are very rare, and occur in the male as well as the female. In fact, the last case of this kind which I treated was in a soldier. These cases are undoubtedly syphilitic and ought to be described as such, but they are very different from the cases which are ordinarily regarded as tertiary syphilitic ulceration. In these cases, on introducing the finger into the bowel, the mucous membrane will be felt to be intact and apparently natural, but the other coats are thickened and indurated over a considerable area and the calibre of the canal materially diminished. This is very different from the cases we are now considering, where the lower part of the rectum is felt to be rough and honey-combed; the diseased portion being surmounted by a contracted fibrous ring, often so small that it will not permit the introduction of the little finger; if, however, the finger can be passed through the stricture, the mucous membrane above will often be found to be comparatively healthy. There is also often evidence around the anus of some unhealthy action going

on, for the skin is frequently found to be much increased and hypertrophied, forming loose folds which hang down and encircle the anus. Fistulæ are also exceedingly common in these cases. These occur from ulceration taking place in the mucous membrane above the stricture, which becomes dilated and thinned from the pressure of the fæces, which are unable to pass through the stricture and become lodged there; fecal matter gets into the ulcers and causes irritation and suppuration, and the matter bursts externally producing a fistula in ano, or into the vagina forming a rectovaginal fistula.

I have little to say regarding the diagnosis. Patients will often present themselves telling you they are suffering from piles, but a little investigation will soon prove to you that this is not so. The history is that the patient first of all began to experience difficulty in defecation, and if the motions were at all constipated, was compelled to strain considerably in order to evacuate the contents of the bowel. The motions may have been noticed to be tape-like and scybalous. Then great pain during defecation with bleeding and discharge of pus, denoting ulceration, supervened. If a digital examination is made, the condition I have already described will be found. A dense fibrous stricture with a rough, uneven, ulcerated surface below. There is profuse discharge of a sanious offensive character and generally intense pain. The patient is apparently healthy at first, but after a time the general health gives way under the pain and exhaustion from loss of blood and discharge.

We now pass on to consider the treatment, and here we are beset with difficulties, for it is often most unsatisfactory. I have told you that I believe the disease is not syphilitic, and therefore I see very little use in giving iodide of potassium and mercury, but I do often prescribe these drugs, more in deference to the opinion of others and as a test that the disease is not specific than for any other reason. The first thing to do is to relieve the contraction and dilate the stricture. This is to be done by the daily introduction of a bougie: an operation simple in itself, but still requiring great care, for the coats of the bowel are often so rotten that the bougie may very easily be pushed through them into the peritoneal cavity and cause the death of the patient. This is by no means an uncommon accident, and therefore the bougie should always be introduced by the surgeon himself, and on no account should the patient be allowed to attempt the introduction of his own bougie. Recognizing the



danger of this proceeding, Christopher Heath recommends using an ordinary old-fashioned dip candle. These candles can hardly do any harm, and, accepting Mr. Heath's recommendation, I always order them when I intrust the introduction to a nurse. Many surgeons recommend smearing the bougie with mercurial ointment, believing in the specific nature of the disease, but for my own part I prefer iodoform ointment of the strength of a drachm to the ounce. After its introduction through the stricture the bougie should be left *in situ* for an hour, or even longer if the patient can bear it; generally the introduction causes very considerable pain, so much sometimes that the patient is unable to submit to it. Under these circumstances, or if the bougie fails to dilate the stricture, as it sometimes does, further operative interference is required, and in these cases I prefer, as a rule, linear proctotomy. The patient having been anæsthetized and placed in the lithotomy position, the forefinger of the left hand is passed through the stricture, a straight blunt-pointed bistoury is then introduced along the finger, and its cutting edge being turned backward, the stricture and coats of the rectum, with the tissues outside the gut, are divided down to the sacrum and coccyx. By making the incision in the middle line backward there is no risk of wounding the peritoneum. There is rarely much hemorrhage, and plugging the wound will always control it. Some surgeons recommend notching the stricture in three or four places with a blunt bistoury, and I have occasionally resorted to this expedient, but on the whole I prefer linear proctotomy as giving the best results. At the same time that these efforts are being made to dilate the stricture the ulcerated surface should be irrigated several times a day with Condyl's fluid or some other antiseptic, and afterwards dusted over with iodoform or, what I think is better, painted over with Whitehead's varnish, which consists of Friar's balsam in which the alcohol is replaced by a saturated solution of iodoform in ether. By this means you may sometimes succeed in dilating the stricture and in getting the ulceration to heal. But you must be prepared for many disappointments, for, as I have said, the treatment is not satisfactory. Perhaps your patient may improve whilst in hospital under this treatment combined with regulated diet and healthier hygienic surroundings; but if she is sent out, she will return in a very short time as bad as ever. Soon the general health begins to fail; she emaciates, gets a nightly temperature, and begins

rapidly to go down-hill. Can anything more be done for her? Yes! we can divert the fæces, so that they no longer have to pass over the ulcerated surface, and then we shall have no difficulty in healing the ulceration and in removing pain and discomfort from the bleeding and discharge. This is best done by performing inguinal colotomy, which is to be preferred to the lumbar operation because we are able to make a much more perfect spur and so entirely prevent the passage of any fecal matter into the lower part of the bowel. This does not cure the stricture, but it removes all source of irritation, and the ulceration speedily heals. The cicatricial tissue thus formed contracts, and it is almost impossible to maintain a channel of any size, so that the patient is condemned to face the alternative of an artificial anus for the rest of her life. This is a frightful affliction, but it is looked upon in a very different light by different individuals. I have had some patients who think very little or nothing of it and go through their daily life with perfect ease and comfort. Notably, I may mention the case of a gentleman for whom I performed colotomy for volvulus of the sigmoid flexure in 1889. He manages his artificial anus admirably; he has perfect control over his fæces; his bowels act once a day in the morning, and for the rest of the day he goes about and enjoys life without inconvenience from his artificial anus. So, again, with the patient to whose case I have already alluded; she suffers no distress or discomfort, but goes about her daily duties, with simply a small pad of carded oakum and a bandage, without suffering any inconvenience. But, on the other hand, there are other sensitive individuals who are in a state of constant horror and disgust at their condition, and I have been told more than once by these patients that they would rather have died than go through what they have to, with an artificial anus. It therefore behooves the surgeon to very carefully explain to the patient the exact state of the case in advising colotomy. If this is done, my contention is that the operation ought to be advised earlier and oftener than it is at present. On the one hand we have an intractable disease which resists all remedies, and which is certain sooner or later to cause death from exhaustion, and on the other we have to face the alternative of a disagreeable and, to some, revolting operation, but with the prospect of restoration to perfect health and prolongation of life. My advice is that in these cases a prolonged effort should be made by the means I have indicated to

heal the ulceration, and should this fail, then the exact state of the case should be fairly laid before the patient and the operation advised. I think there is one other point which should be present in the surgeon's mind in coming to a decision as to advising an early operation, and that is that, even supposing he does succeed in healing the ulceration, he will still have to deal with the stricture which will result and which he cannot cure, so that the patient will have to face the fact that she will have to submit to the passage of bougies for the rest of her life.

## INSTRUMENTAL ORCHITIS.

CLINICAL LECTURE DELIVERED AT THE TREMONT DISPENSARY, BOSTON.

BY CHARLES GREENE CUMSTON, B.M.S., M.D.,

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Society of France, of the Pathological  
Society of Brussels, etc.

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GENTLEMEN,—This patient is a man of sixty-seven years, and as you see he presents the outward appearances of a good constitution. He follows the trade of a tailor, and little or nothing of importance can be found in his personal antecedents. He had one attack of gonorrhœa at the age of twenty-seven, which was cured at the end of six weeks and never gave rise to chronic posterior urethritis.

For the past two years he has had difficulty in passing his urine, and some ten days ago he had a retention which necessitated the withdrawal of the urine by a catheter, which was done by a physician who was summoned at that time. But he comes to us to-day not for the difficulty of passing his water, but for an inflammation of the left testicle, which, according to the patient's story, began to manifest itself day before yesterday.

As you see, the left side of the scrotum is considerably enlarged, the skin is red and tense, and by palpation the testicle is found to be rather hard, not very painful, and somewhat enlarged. The spermatic cord and epididymis are apparently normal. Rectal examination shows us that there is an enlargement of the right and left prostatic lobes. The temperature is  $38.1^{\circ}$  C. The urine is alkaline in reaction and contains a few leucocytes, but no red blood-corpuscles and only a few bladder-cells.

We have here a case of instrumental orchitis, and with your permission I would like to consider this subject, as it has an importance

that is of no little weight to the general practitioner. Many and various are the factors which may give rise to an orchitis; from direct traumatism of the gland and the different affections of the urethra to diseases like typhoid fever, mumps, and small-pox, which on account of their infectious nature quite often give rise to special localizations. Gonorrhœa is without doubt the commonest factor, but changes in the prostate gland and bladder, more especially trigonitis, tuberculosis or carcinoma of the bladder, calculi, and the passage of either metal or rubber instruments into the urethra or bladder, are frequent causes. Gout, and less frequently rheumatism, give rise to it.

We will only consider in this lecture the orchites produced by the passage of instruments. Velpeau said in his lectures that healthy and strong persons who underwent a simple catheterization unattended by pain or difficulty are afterwards suddenly taken with chills, fever, swelling of the knees, delirium, and that death was often the result. The great French surgeon was alluding to those very serious complications which occasionally complicate the most careful and antiseptic catheterization, which we cannot foresee nor prevent; but such severe symptoms are fortunately very infrequent at the present time. There are, however, certain ones that are quite frequently met with in practice, and although they do not have the same gravity as those already mentioned, they still have their importance. These complications are fever, hemorrhage, inflammation of the prostate, dysuria, and orchitis.

As I have already pointed out, the latter complication may be produced by either metal or rubber instruments introduced into the urethra, but their use is varied. The surgeon may employ them simply to explore the canal of the urethra, for the treatment of stricture by dilatation, internal urethrotomy, instillations in the posterior urethra for chronic urethritis, drawing off the urine in cases of retention, as in the patient just shown, or for making a diagnosis of calculus, tumor of the bladder, or irrigation of the organ. Consequently the organs passed over by the instrument may be normal or the seat of some pathological process. The mucous membrane of the urethra may be nearly normal or be the seat of an anterior inflammation, but an orchitis will appear indifferently in any of these conditions.

It is, however, most important to find out if certain changes of

the genito-urinary system do not more particularly predispose the patient, and to demonstrate if possible during what affection this instrumental orchitis is the most frequent. Unfortunately we are quite in the dark, and in order to come to a conclusion a large number of cases of a given affection of the bladder, prostate gland, or urethra must be collected and analyzed, after which some conclusion as to its pathogenesis might be drawn. Pilven found that in one hundred and eighty-eight cases of vesical calculi who had undergone exploratory catheterization or lithotripsy in the service of Professor Guyon, thirteen presented an orchitis, and he believes that this complication was due to the exploratory catheterization and to sounds left in the bladder for constant drainage. He also thinks that the operation of lithotripsy must be taken into consideration as giving rise to a morbid predisposition.

Velpeau considered orchitis as a very frequent complication of lithotripsy, and Civiale was of a similar opinion, while Philips considered it as present in about two-thirds of the cases of this operation. Thompson believes that orchitis is very infrequently met with after lithotripsy, and only found one case out of two hundred and thirty-four operations.

Pilven also examined the records of one hundred and fifty-two cases of stricture of the urethra following gonorrhoea, treated either by dilatation or urethrotomy, and found that six times there was an orchitis as a complication. In two cases there was suppuration. Dilatation with bougies and catheters left *in situ* appeared to be the cause. Raybard says that dilatation exposes patients to all inflammatory action that is produced by the introduction of a foreign body into the urethra, but believes that gradual dilatation is more frequently the cause of orchitis than the rapid method. Internal urethrotomy may also give rise to various complications, but its part is trifling in the etiology of orchitis if we admit that the action of the instrument should make itself felt in the posterior urethra, because strictures are always situated higher up than the prostatic region. Tillaux only found one case in a large number of urethrotomies, and this occurred after passage of tin sounds ulterior to the operation.

In old people with hypertrophy of the prostate a retention of urine frequently takes place, which necessitates the introduction of the catheter, as in our patient of this morning, and out of forty-four

collected cases orchitis occurred four times. Of seventeen cases of tubercular cystitis that necessitated the passage of the catheter, orchitis was noted in three; two of them, aged respectively fifty-three and sixty years, had a simple epididymitis, while the third had three successive orchites after three different catheterizations.

In eighty-two cases of chronic posterior urethritis Pilven found an orchitis in two patients aged forty-two and thirty years. The latter had a double orchitis after the simple removal of a calculus. The same writer found one case of orchitis out of twenty-four cases of lithotomy, and this complication occurred after exploration of the bladder with sounds.

From what has been said it may be concluded that it is principally the action of the instruments on the mucous membrane of the urethra that must be taken into consideration independently of the disease necessitating their introduction, but we must not lose sight of the fact that this inflammatory complication of the testicle is more frequently met with in men advanced in years. As we know, the mucous membrane of the urethra appears to have a special susceptibility in elderly men, and it becomes more vascular and delicate than in adults, and if there is an hypertrophy of the prostate the urethra is deformed in that region. An instrument may with difficulty be passed, and for this reason the chances of an irritation of the prostatic region are increased, and it is just at this point that all those conditions favorable to the production of an inflammation of the testicle are to be found. The anatomical structure of the urethra becomes more complicated the nearer we approach the bladder, and at its most distant part we find, beside the microscopical orifices of the mucous glands, the orifices of the excretory canals of the prostate, the largest of which are to be found on the lower aspect of the urethra. Then we have the vas deferens, which open on each side of the veru montanum and connect directly with the epididymis, and the testicle with the urethra by the continuity of their mucous membrane with that of the urethra.

To this anatomical arrangement, which opens up an easy road for inflammation, we may add the curved direction of the urethra, on account of which the instruments press against its posterior wall. As to the projection of the veru montanum, Guyon believes that it is no obstacle to the passage of sounds, but it must not be harshly handled on account of the neighboring canals of ejaculation. If

there is no pathological change in the mucosa of the urethra at the beginning, it will not be long before one occurs under the influence of the introduction of instruments, because all cases of stricture submitted to dilatation, all patients undergoing operative treatment, and those wearing a catheter in the canal have a certain amount of inflammation of the urethral mucosa, and there is often a profuse discharge.

There is without doubt a relationship between orchitis and the condition of the prostate and the openings of the vas deferens on account of their anatomical situation, as was pointed out by Civiale many years ago. "In some subjects a pathological condition in these organs predisposes them to inflammation, and the testicles may be the seat of this process when an instrument acts forcibly on that part of the urethra near the neck of the bladder or when by rough handling lesions are produced and keep up an irritation of the neck of the bladder, and especially when, on account of an old stricture, the prostatic region of the urethra has been seriously injured and the prostate is invaded by a permanent morbid condition." The morbid condition of the posterior urethra may be the means of producing an orchitis in many instances, but it is far more frequent to witness an orchitis following a catheterization, as if the instrument acted by producing an acute inflammatory attack in those parts already the seat of a chronic inflammation.

The rough or careless use of instruments cannot be accused of directly producing an orchitis, and although by this means false routes and abrasions of the mucosa may result, still, if an orchitis appears, this complication must be considered as due to the particular susceptibility of the patient and to the pre-existing condition of his urethra. The most careful catheterization will not prevent the occurrence of orchitis, and it is not infrequent to observe this complication after a simple introduction of a soft gum catheter in a normal urethra.

Now, what is the way in which instrumental orchitis takes place? In order to explain this we must consider the various theories put forth regarding gonorrhœal inflammation of the testicle, although the difference is great between the instrumental and gonorrhœal forms. In gonorrhœa the inflammatory process remains stationed in that part of the urethra situated above the membranous portion and the testicle is free from danger; but if the process extends



farther down the canal, either by direct tissue extension or by forcing the pus backward by too powerful injections, the gonococcus will penetrate the orifices of the vas deferens and invade the epididymis and testicle. Such is the starting-point of gonorrhœal orchitis, and such is also the starting-point of an orchitis when an instrument acts on the prostatic region of the urethra, on account of a certain amount of irritation that it is almost sure to start up. Gosselin reports a case of orchitis which developed under the influence of a urethritis produced by the passage of catheters, and Guyon upholds that any instrumental manoeuvre in the canal sets up an inflammation, and we must also remember that behind strictures there is most always a chronic inflammation present.

Hardy and other observers believe that the mechanism is the same in both instrumental and specific orchitis, but, although they admit that an irritation of the vas deferens is necessary, they differ on other points. Some think that the testicle becomes inflamed because the inflammation of the urethra extends to it by the vas deferens, or, in other words, by continuity of the tissues. Others put forward metastasis, while angioleucitis or a phlebitis of the veins of the cord have been considered as the means of propagation to the testicle.

When we have the chance of watching the affection from the beginning, after catheterization, we notice that the cord becomes hard and painful, and then becomes tumefied; after this there is pain and swelling in the epididymis and testicle. At autopsy inflammatory lesions are found extending from the prostatic region to the epididymis along the vas deferens and the seminal vesicles. All these facts would lead us to suppose that extension of the inflammation takes place by the spermatic cord. The introduction of bougies through a stricture and their sojourn in the deep urethra produce an inflammatory process which may invade the vas deferens extending down to the epididymis, just as it may invade the prostatic glands, Cowper's glands, or the bladder, and finally may extend up the ureters and reach the kidney.

The symptoms of instrumental orchitis are very similar to those occurring in the gonorrhœal type when the instrumental type is at all severe, but certain variations in the progress and termination are alone sufficient for us to make a distinction. Often after a catheterization or other instrumental work in the urethra, after a few days,

or even suddenly in a patient obliged to pass a catheter regularly, a slight pain is noticed in the spermatic cord and a feeling of dragging are the early symptoms of an orchitis. But generally the commencement is more insidious, and it is by chance, so to speak, that by putting his hand to the parts the patient notices that his testicle is enlarged, and at the same time that there is more or less pain when the organ is pressed, and that this pain extends up the spermatic cord, which may also be found enlarged.

From this time on the increase in size of the testicle goes on rapidly. The scrotum becomes tumefied, shiny, red, and painful, while the local temperature is quite elevated. The epididymis will be found to be twice or three times its normal size, and is hard and painful. It may be so much enlarged that it covers the testicle like a hat and completely covers the gland. The testicle, however, does not necessarily escape the inflammatory process, and if it is relatively smaller than the epididymis in many cases, it may, nevertheless, be two or three times its normal size.

When the parenchyma of the gland is thus attacked the intensity of the pain increases, a fact easily explained when we remember how the tunica albuginea resists the swelling of the gland due to the inflammation. We frequently see the vaginal tunic become the seat of a serous collection, usually not of any great amount, but which will put the serous membrane on the stretch if by chance the amount of liquid should become considerable, causing such severe suffering that withdrawal of the fluid becomes absolutely necessary.

Some cases, in which the epididymis and testicle participate equally in the inflammation, will present a homogeneous mass, and it is impossible to distinguish one organ from the other, and they can only be made out separately after resolution has commenced. At the same time that the testicle and its envelopes are the seat of the inflammation, the symptoms in the spermatic cord also increase, but usually the process disappears more rapidly than that of the testicle. Although the swelling may subside in the cord it may be increased in size for quite a long time, but no pain will be experienced there, although in the beginning there may have been some pain extending to the lumbar region.

After a few days the inflammatory process will have reached its height, remaining stationary for some days, and then progressively diminishes. When the orchitis appears the temperature will be up,

ranging between  $38^{\circ}$  to  $39^{\circ}$  C., and will remain at this point, with remissions, during the period of increase. Gastric disturbances, anorexia, sometimes constipation, may complicate the local condition, but they are very slight, and may even pass by unnoticed when the orchitis is not severe.

Philips, Reliquet, and others say that an instrumental orchitis rapidly reaches its height, and that the first stage is shorter than gonorrhœal orchitis, and that in the latter affection the inflammatory symptoms are far more pronounced. When resolution occurs, the second period is, on the contrary, longer in the instrumental type, and may last for a month or more. Generally the progress of instrumental orchitis is not as uniform or as regular as in gonorrhœa. In the latter type the tumefaction and pain in the spermatic cord and epididymis become rapidly severe and then these symptoms regress, leaving behind them a more or less extensive induration, while in the case of instrumental orchitis only a temporary and almost indolent swelling of the spermatic cord and epididymis occurs, and which disappears usually by simply stopping the use of the instruments and keeping the patient quiet.

But we will meet with some cases of instrumental orchitis which do not progress so favorably, especially is this true of old men, and then we find the skin of the scrotum red, hot, adherent to the underlying parts, in which case suppuration is to be feared. The general symptoms vary in severity, but are more marked than in the slighter forms, and are present usually when the spermatic cord and seminal vesicles are involved. There are fever, vomiting, hiccough, and chills, and when these symptoms do occur beware of the kidneys; look for any pathological condition in the urine which may put you on the track of some renal complication, because if one is found it may explain the serious condition of your patient.

Underneath the inflamed scrotum the deeper structures will be found tense, hard, and enlarged, and both on account of the inflammatory thickening of the scrotum, epididymis, and testicle, it is very difficult to decide which is the principal seat of the affection. When the vaginal tunic is distended it is impossible to detect the starting-point of the suppuration. When a suppurative process takes place in the vaginal tunic or epididymis, or in both at the same time, a rather extensive point of fluctuation may be found over the anterior aspect of the scrotum, or an abscess may form in the tissues

of the scrotum itself. Incision should at once be made before the tissues have become thinned.

If suppuration takes place in the parenchyma of the testicle fluctuation is long in showing itself externally, and in some instances it may be completely wanting. Very acute deep-seated pains should be your indication for incising the tunica albuginea, which will put a stop to these symptoms of strangulation. As a general rule when by incision we have given the pus an exit, both local and general symptoms become markedly improved. Suppuration, which in these cases usually develops rapidly, lasts for several days, and varying quantities of pus will be discharged through the incision, and when it comes from the testicle, seminiferous canalicules will be found projecting through the wound made in the tunica albuginea, forming a sort of fungous growth. This will also happen if you are not careful to replace the canalicules under their enveloping membrane, and they will be eliminated by a necrobiotic process. Little by little under proper treatment the secretion of pus will subside and the wound heal, taking in all some fifteen days, perhaps more.

To sum up, we may say that instrumental orchitis takes on two types, which are of equal frequency: *first, a subacute type, ending by resolution of the process; second, an acute type, usually terminating in suppuration.*

In both, the testicle, epididymis, and vaginal tunic may be all included in the process, but in the majority of cases it is the epididymis that is alone the seat of the inflammation, although it has been asserted by Mickaniewski that suppuration of the epididymis has never been observed. I think, on the contrary, that this is a frequent occurrence, and reported cases and some few that I have myself seen lead me to be able to positively deny the above statement as incorrect.

The epididymitis is usually complicated by a fluid collection in the vaginal tunic in quite a number of cases, but in many the amount present is not enough to give rise to trouble.

Generally the acute type of instrumental orchitis ending in suppuration is of infrequent occurrence, but when you are called to attend these cases the possibility of such an event must be borne in mind.

The diagnosis of instrumental orchitis is to be made from the history of the case, because its symptoms and progress are not always

sufficiently characteristic to allow one to differentiate it from other affections of similar type. The absence of any traumatism, gonorrhœa, or infectious disease will permit you to eliminate them as an etiological factor. As to the various varieties of urethral orchites, they arise spontaneously under the influence of some lesion of the genito-urinary organs that will be discovered by a carefully conducted exploration.

Cancerous, tuberculous, or syphilitic<sup>1</sup> epididymitis have distinct characters which will give you a key to the correct diagnosis, and in cases of acute genito-urinary tuberculosis, rectal examination will usually reveal lesions in the prostate gland.

The relations of the cause and effect will be most evident when shortly after the introduction of instruments into the bladder or urethra an inflammatory process appears in the testicle.

The prognosis of instrumental orchitis depends entirely on the intensity of the inflammatory symptoms. When the affection is slight, as in the case of this morning's patient, it will disappear without leaving any trace behind and the gland will return to its normal condition and functions. It is different, however, when the inflammation has been severe, when the testicle, and especially the epididymis, have been the seat of much tumefaction with a catarrhal inflammation of their canals. The plastic formation in this case may be so considerable that absorption takes place very slowly, and may even leave behind a certain amount of induration. The inflammatory process may affect the intercanalicular connective tissue, producing embryonic neoformed tissue; then it in turn acts on the sub-epithelial connective tissue and causes a sclerosis of the canals, some undergoing dilatation, while others become atrophied. Thus when the affection becomes chronic it produces sclerosis and atrophy of the tail of the epididymis, at which point these lesions are most frequently met with.

It must, however, be said that atrophy is infrequent when the orchitis has not been complicated by suppuration. It is rarely met with after gonorrhœal orchitis. The tumefaction of the epididymis with induration of its tail is, on the other hand, quite frequent. The testicle, which is less frequently the seat of the process and which is usually less severely attacked when it is invaded by the inflamma-

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<sup>1</sup> Cumston, "Tertiary Syphilitic Epididymitis," *Annals of Surgery*, March, 1897.

tion, will most always recover its normal size and consistency, but the prognosis is none the less bad, for the reason that the sperm may be prevented from flowing out, because if the calibre of the epididymis is obstructed the seminal fluid is retained. Consequently the function of the organ is abolished, but it is evident that the patient will only be sterile when both testicles have been invaded by the inflammation.

The focus of induration in the epididymis is usually indolent and gives the patient no trouble, but over-exercise or pressure on the organ may sometimes cause a tenderness, which soon passes off with rest. The absorption of the inflammatory exudate takes some time to be complete, and may last for months or even years, and for lifetime in cases of gonorrhœal orchitis.

It has even been upheld that the remains of the inflammatory process were the starting-point of various types of degeneration, such as syphilis, tuberculosis, or cancer, but if such does happen, the testicle acts only as a *locus minoris resistenciæ*. Relapses are frequent, and after having had one attack of orchitis the patient should be instructed to avoid anything that could cause it to return.

When the epididymis or the testicle suppurate, the prognosis partly depends on the rapidity with which surgical interference is resorted to. There are two circumstances to be feared in this case, especially when the pus becomes infiltrated in the parenchyma of the organ instead of forming a circumscribed collection; in the first place, if it remains for any length of time it will be pretty sure to destroy the delicate structures, and when it forces its exit outward, either spontaneously or through an incision performed too late, the seminiferous tubes are extruded from the opening when the glandular parenchyma is affected, and your patient will run the risk of total elimination taking place with a consequent entire loss of the organ. But a spontaneous opening does not always occur, and the pus, whether infiltrated or collected, may become partly absorbed, while its solid constituents alone remain indefinitely, forming an indolent and hard mass.

Now, if you do incise the abscess of the epididymis in time or open the tunica albuginea, the result of the suppurative process will be most likely not very serious, and a cure will take place promptly. The peritesticular inflammation is relatively slight if the vaginal tunic is tapped early, but if you neglect doing this and the serous

membrane becomes lined with neoformed membranes, which become organized and thicken the sac, the ultimate result will in all probability be a fistula.

As a very exceptional complication I might mention the extension of the inflammatory process up the vas deferens to the peritoneum. Remember that an instrumental orchitis, although not serious, generally speaking, should be a warning to you as to the susceptibility of the urethra of your patient, and should make you fear the easy occurrence of more serious phenomena.

Let us now consider the treatment of this affection. Those measures that have been employed for gonorrhœal orchitis will find their place in the treatment of the instrumental form, for although the etiology is different, as well as its clinical picture, the fundamental lesion is the same in both cases. We should endeavor to prevent the congestion of the testicle and to combat the inflammation when it is once declared. The next point is to avoid suppuration, but if it does occur in spite of our treatment, a rapid exit must be given to the pus before perforation occurs and the gland has undergone too much destruction.

To prevent congestion of the testicle is not easy, and you must always bear in mind this possibility when you are passing urethral instruments for the first time in a patient; you must take all necessary precautions, which, although they are not a perfect guarantee of prevention, will at any rate diminish the risk.

These precautions are the exhibition of sulphate of quinine combined with salol or urotropin for two or three days previous to the exploration and a perfect asepsis of hands, instruments, and urethra. To render the easy and rapid passage of instruments I would recommend you to employ one of the following ointments that I have been in the habit of using for some time:

R Cocaine hydrochlor.,  
Creoline, ʒʒ grs. vi;  
Lanolin, ʒi. M.  
Sig.—For external use.

R Eucain. B, ʒi;  
Sanoform, ʒi;  
Vaselin. liq., ʒi.  
Sig.—For external use.

This formula is sterilized at 100° C. for one hour, as none of the ingredients are decomposed by a high temperature.

After the exploration it is well to order your patient to keep his bed for the remainder of the day. Do not forget to attend to the bowels and have them moved freely the night before with salts.

When your prophylactic measures have failed to prevent the complication from occurring and the epididymis and testicle are the seat of swelling and pain, we must try to diminish the intensity of the process, calm the pain, and prevent suppuration from taking place. It is true that in many cases the affection will not continue beyond the stage of hyperæmia, and to dispel this rest in bed and ceasing all instrumentation in the canal of the urethra will generally suffice. If there is much pain, the scrotum must be elevated by means of a soft towel.

Internally, pilocarpine, at the dose of one centigramme subcutaneously, twice daily, has been well spoken of, and I can thoroughly recommend the administration of pulsatilla, which I have used on several occasions with good results, as follows:

R Tinct. pulsatillæ, gtt. xxx;  
Syr. sacchar.,  
Aq. menth. pip., aa ℥ii. M.  
Sig.—A dessertspoonful every hour.

The application of medicines to the scrotum is also to be combined with rest and internal treatment, and foremost of all I commend to your notice the simple mercurial ointment. American physicians appear to be afraid of mercury generally, but when used with care and judgment it is one of the most potent articles of the pharmacopœia. When I employ it for inflammatory processes that are localized, as is the case with orchitis, I combine it with some form of the iodides and belladonna ointment, and the results that it has given me allow of my giving it my highest recommendation. I formulate as follows:

R Ung. hydrarg.,  
Ung. belladonnæ, aa ℥iv;  
Iodol, ℥ii. M.  
Fiat ung. et div. in chart. cerat. No. xii.  
Sig.—Apply one package to the parts morning and night.

In prescribing mercurial or other ointments do not tell your patient to apply a piece the size of a pea, etc., but divide the mass



into waxed paper packages as in the above formula, and then you will know exactly how much of the active principle is being used in twenty-four hours.

Another very good preparation that has also given me good results is a twenty-five-per-cent. ointment of ichthyol, but I cannot say that it is in any way superior to the mercurial ointment. Ichthyol may be combined with the iodide of lead and chloride of ammonium as follows:

R Ichthyol,  
Plumbi iodid., aa ℥ii;  
Ammon. chlorid., ℥i;  
Ung. simpl., ℥i, ℥vii. M.  
Fiat ung.

Sig.—Apply to the parts thrice daily.

You must also attend to the intestinal functions, and you will commit no error if you produce two or more watery stools a day, as it certainly will have an excellent derivative effect, while if there is intense pain the following may be ordered:

R Chloral. hydrat.,  
Potassii bromid., aa grs. xlv;  
Syr. cort. aurant., ℥i;  
Aq. dest., q. s. ad ℥iv. M.

Sig.—Take a tablespoonful; repeat in two hours if necessary.

When a considerable amount of fluid has become collected in the vaginal tunic, producing tension and pain, puncture should be made and the liquid withdrawn, but this should not be done unless there is a considerable quantity of serous fluid present.

When the inflammatory stage has passed by and is succeeded by induration, you must try to bring about absorption by the internal administration of small doses of the iodide of potassium, but it must be watched, for if given for too long a period it may be the cause of atrophy of the testicle.

Now if suppuration does occur, exit must be given to the pus just as soon as fluctuation can be detected, and remember that an early incision will greatly diminish the gravity of the prognosis. But as it is difficult to make out surely the origin of the suppuration, when the vaginal tunic is distended or when the abscess is pointing under the skin, you should incise layer by layer so as to avoid cutting

the testicle, which if healthy will be kept from injury by a grooved director. The incision must be made large enough so as to secure perfect drainage and prevent the formation of a fistula.

When the testicle itself is the seat of suppuration and you have incised the tunica albuginea, the seminiferous tubes will escape with the pus. In order to prevent their complete exit they must be pushed back in the scrotum, and what cannot be reduced must be removed by scissors, and thus you will be able to preserve part of the gland, if not the whole, and its functions will not be lost. Antiseptic dressings and irrigations frequently repeated will bring the case to a happy cure.

## THE X-RAYS IN SURGERY.

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WHEN the X-rays were first used in surgical cases surgeons were satisfied with a skiagram showing some bone deformity or the mere presence of a foreign body, such as a fragment of needle, bullet, etc., but as the novelty of even such a result being obtained passed away and the use of these rays became more general, it was felt that some method of precise measurement was essential.

It so happens that the X-rays lend themselves in a peculiar way to enable such precise measurements to be made. They cannot be twisted out of their linear path,—in other words, they cannot be refracted,—and they refuse to travel excepting in one unbroken, straight line from their point of origin (a small surface on the anode of a Crookes tube). Now this property of being unrefrangible, which is such a puzzle to the physicist, is precisely the one which is so useful to the surgeon,—for this reason, that when a skiagram is obtained we know it is produced by rays which have come from a point and travelled in straight lines through the body to the photographic plate. Thus we get a geometrical projection on one plane of a variety of structures occupying different planes.

The problem that had to be solved was how to localize—how to find out, for example—the exact size and position of a fragment of needle embedded in the tissues.

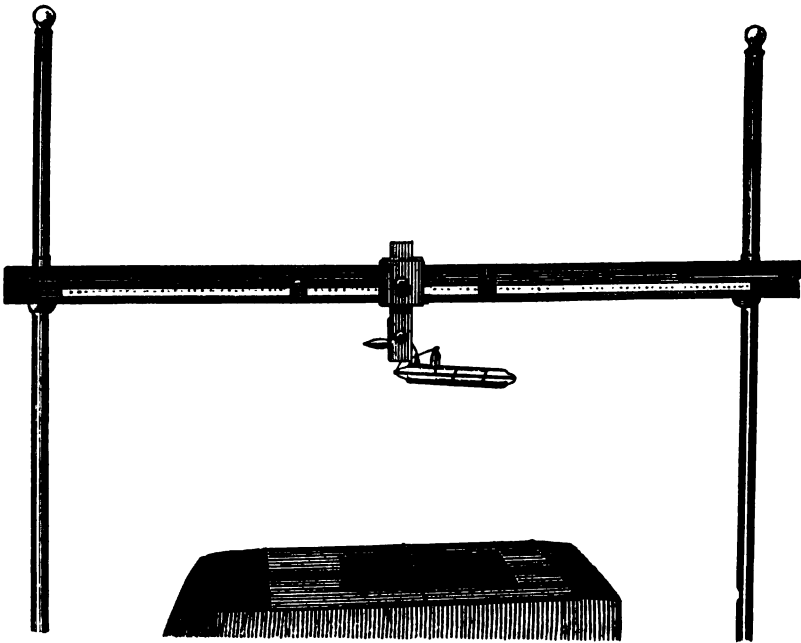
I have devised a method of solving the problem, and it has yielded such excellent practical results that I venture to bring it under your notice. All attempts at localization had necessarily to be based upon taking two or more skiagrams from different points of view by moving the Crookes tube to different positions, and the interpretation of the skiagrams so obtained involved the use of mathematical formulæ or geometrical drawing to scale.

By the method I have devised, and am about to describe, precise results are obtained from beginning to end by means of measurements alone. While the method is simple, it is yet based upon correct geometrical principles.

I obtain the three coördinates of any desired point of the foreign body, and the way this is done is as follows:

The Crookes tube is fixed in a holder which slides along a horizontal bar. (See Fig. 1.) On the front of the bar a scale is fixed

FIG. 1.



Skilagraphic localizing apparatus.

with the  $0^\circ$  in the middle, which for greater convenience may be graduated in centimetres and millimetres. On the top of the bar there is a small spirit-level. Two brass rods support the cross-bar at its ends and enable it to be raised or lowered as desired. One of the edges of the tube-holder is placed at  $0^\circ$  on the scale, and beneath it on a table is placed a large sheet of vulcanite with two wires tightly stretched, and crossing each other at right angles at its middle point. A small plumb-line is now dropped from the middle of the anode of the Crookes tube, or, rather, from a point on the glass as near to it as possible, and the sheet of vulcanite is so adjusted that

the point where the wires cross is vertically below the anode. Further, one of the cross-wires must be accurately parallel to the horizontal bar. These adjustments having been made, the vulcanite is fixed to the table by means of drawing-pins.

The distance of the anode from the point where the wires cross is measured and noted. Two skiagrams have now to be taken, the tube in each case being displaced a measured distance to opposite sides of the  $0^\circ$  on the scale. What this displacement is to be does not matter much, but when it has been decided, two small clips (see Fig. 1) are brought from either side to the points on the scale and then clamped. This enables the tube-holder to be displaced to either side up to the clip as a limit. A note of the displacement is kept.

The holder is then displaced to one side, when a photographic plate in an opaque envelope is placed beneath the cross-wires, and these are brushed lightly over with some ink, so as to leave their mark on the patient's skin. The patient now places the part to be photographed on the plate, being careful not to move, once the skin has come down on to the wires.

An exposure is given, and then the tube is displaced over to the other side of the  $0^\circ$  and another exposure given on the same plate or on a different one. A small opaque object should be placed on one corner of the plate, and a mark made on the patient's skin nearest this object. This is simply to remind one of the position of the patient in relation to the skiagram.

This completes the taking of the skiagrams. I must now call your special attention to the significance of the arrangements I have described.

In the first place, we have the photographic plate that represents one plane, viz., the horizontal. The two cross-wires represent two vertical planes which are at right angles to each other as well as to the surface of the plate.

In working out the results, I have devised a simple apparatus shown in Fig. 2 which may be called the "*cross-thread localizer*." This is designed so that the negative (if both exposures were made on one plate) or a tracing from the negatives (if two plates were used) should be placed under precisely the same conditions as existed when it was being produced.

Fig. 2 shows a horizontal scale with the  $0^\circ$  in the middle. (This

corresponds to the horizontal bar in Fig. 1.) Beneath it there is a horizontal sheet of plate glass with two lines marked on its surface with a diamond.

They cross each other at right angles at the middle point, and the  $0^\circ$  of the scale is exactly vertical to the point where the lines cross, and, further, the edge of the scale is in an exact plane with the line on the glass that runs right and left. This corresponds to the vulcanite with the cross-wires.

A mirror is placed below simply to reflect the light upward and render the negative visible by transmitted light when it is laid upon the glass horizontal stage.

Suppose that the negative we have to deal with was taken when the anode of the Crookes tube at  $0^\circ$  was say twenty centimetres above the cross-wires. I would then place the  $0^\circ$  of the sliding scale twenty centimetres above the point where the lines cross on the glass stage.

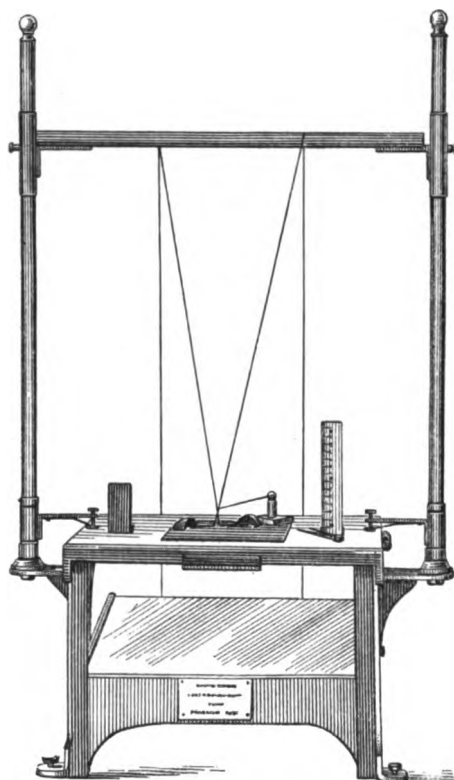
The negative, after being fixed and washed slightly, is placed upon the stage in such a way that the white cross produced by the wires exactly coincides with the cross on the glass stage, and, further, the mark on one of its corners enables it to be placed in relation to the scale, etc., as it was when being taken. All that is necessary now is to trace the paths of the X-rays which produced the double shadows, and I do this by means of two fine silk threads attached to two fine needles. The other ends of the threads have small lead weights attached to keep the threads taut, and the needles are weighted with lead. (See Fig. 2.) For convenience the scale is notched opposite the graduating marks, so that the threads can be placed in any desired notch.

Suppose that in taking the negative the anode of the Crookes tube had been displaced to say six centimetres each side of  $0^\circ$ . Then these threads would be placed in the notch of the scale opposite six centimetres on either side of the  $0^\circ$ .

Matters being thus adjusted, we find the negative will show two shadows of the foreign body. The one to the right will, of course, have been produced when the Crookes tube was displaced to the left, and so we take the needle with the thread attached coming from the left side and place on any desired part of the shadow on the negative. We place the thread coming from the right side in the same way on the corresponding point of the left shadow.

Now these threads represent the path of the X-rays that actually produced these shadows, and so the point where the threads cross represents the position in space actually occupied by that point of the foreign body.

FIG. 2.



Cross-thread localizer.

We now proceed to measure the three coördinates of this point where the threads cross. First, we take the vertical distance from the negative, which represents the depth of the foreign body below the surface of the skin lying nearest the plate. We then place a rectangular square on the edge of one of the wire shadows and measure the vertical distance from this plane by means of a pair of compasses. The same is done with the other wire shadow, and we then have the three coördinates, which we note down (see Fig. 3) as follows:

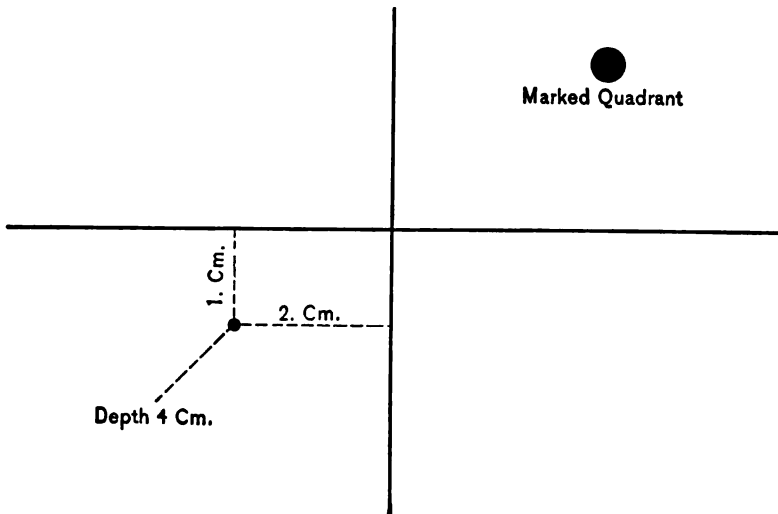
The patient has the mark of the cross-wires on his skin. We

measure the two coördinates on the surface of his skin, and where they meet fixes the point below which vertically lies the foreign body at the depth given by the third coördinate.

The method, while not easy to describe, is yet easy to carry out, and gives results which cannot fail to be accurate, provided due care is exercised in the necessary adjustments.

To show its possibilities, I may mention that I have, with some slight modifications, applied it to the localization of foreign bodies in the eyeball. In twenty cases it was done with great accuracy, as was subsequently proved by the extraction of the foreign body by

FIG. 8.



Method of recording measurements of the three coördinates of a foreign body as estimated by the cross-thread localizer.

the electro-magnet, and still better demonstrated in some of the cases in which the eyeball had to be enucleated.

In ophthalmic cases it is necessary to fix the head, and to get the patient to look at an object during exposure. Further, a small piece of lead wire is placed on the lower eyelid in a known position to the eyeball. Two plates are used, and the exposure necessary is about ninety seconds for each. The tracing from the negatives is dealt with on the cross-thread localizer as I have already described.

The three coördinates of the point of lead wire are measured, then the three coördinates of the foreign body are measured, and it



is, of course, very simple to find from these the three minor coördinates which show the relation between the position of the foreign body and the known position of the point of the lead wire. With care, the position can be ascertained to the one-fiftieth part of an inch.

Any one who follows the principles underlying my method can construct very simple apparatus to carry it out. But, of course, for fine work, such as a foreign body in eyeball or orbit, a well-made, rigid apparatus is necessary. Messrs. Curry & Paxton, London, have made the apparatus for me.

## CIRRHOSIS OF THE LIVER.

CLINICAL LECTURE DELIVERED AT GUY'S HOSPITAL, LONDON.

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Physician to the Hospital.

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GENTLEMEN,—We have had many opportunities lately of studying cirrhosis, but in the short time of an hour we can only consider two cases. The first that I propose to take is the following:

CASE I.—Eliza P., aged forty-one, was admitted into Mary Ward on May 29, 1897. She had been previously admitted on May 12, and she then sought admission for swelling of the abdomen and legs. She had been a hard drinker. For the preceding months her feet had swelled, and for the last six months the abdomen had been enlarged. For some time she had suffered from morning vomiting, and lately she had become slightly jaundiced and noticed that her urine was scanty, high colored, and contained a red deposit. The liver could be felt to be hard and nodular. The day after admission the abdomen was tapped and fifteen pints of ascitic fluid were withdrawn. After this she only remained in the hospital a few days, but even in this time the abdomen had almost regained its former size. On May 29 her new report states that she was a woman who looked older than her age, was rather wasted, her face was red, and she was slightly jaundiced. Her legs and tongue were tremulous, and she was distinctly drowsy. Her abdomen was much distended, being forty-four inches in circumference; the umbilicus was prominent; the superficial veins on the abdominal wall were much distended, and the blood flowed in them from below upward. There was evidently much fluid in the abdomen, for it was dull in the flanks, but the dulness there disappeared if the patient lay on her opposite side, and it was easy to obtain a thrill. The liver could not be felt, but the spleen could be made out on dipping. The pulse was 102, and there was a hæmic

murmur in the pulmonary area; otherwise the circulatory system appeared normal. On examining the chest, we found that there were a few rhonchi to be heard in front, and many rhonchi and râles could be heard behind, especially at the base. Here the entry of air was bad and the percussion note was dull. The patient's legs were œdematous. Her urine contained no albumen; its specific gravity was 1010. She was ordered some compound jalap powder at night, to be followed by some sulphate of magnesium in the morning, and we also gave her large doses of copaiba resin and restricted the fluids she drank. On June 4 the abdomen was tapped, when twenty-one pints of fluid containing albumen were drawn off. The next day she was still drowsy. The liver could be easily felt, the margin being in the nipple line six inches below the ribs, and the surface felt hard and rough. The only things further to say about this patient are that her jaundice became deeper and deeper, her weakness increased, and she became more and more drowsy, until at last she could hardly be roused. She died on June 22. At the post-mortem examination it was found, as we expected, that she had cirrhosis of the liver. The organ weighed seventy-four ounces, its surface was nodular, and its edge irregular. It was very tough on section, showing small bright yellow areas surrounded by white fibrous tissue. The cirrhosis was both unilobular and multilobular.

CASE II.—This patient is fifty years old, and she now lies in Miriam Ward. She has drunk a considerable amount of beer and spirits. Three months ago her feet began to swell, and three weeks ago her abdomen began to increase in size. She has vomited occasionally during the last three months, and has been much troubled with piles. She has never been jaundiced. If you will go into the ward you will see she has wasted, and that she has dilated venules on her cheeks and nose. The abdomen gives the same indications of fluid within it that we noticed in the first case. The liver cannot be felt. The heart and lungs are healthy. The urine contains a trace of albumen.

Now in both these cases the diagnosis made at the bedside was that the patient was suffering from cirrhosis of the liver, and it is therefore necessary that before we go any further you should have a clear idea of what we meant. We meant that whenever the patient died we should, even with the naked eye, be able to make out that the fibrous tissue of the liver was, uniformly throughout the

organ, greatly in excess of the amount found in healthy livers. Because of this the cirrhotic liver is very hard, and usually by the time the patient dies this abnormal fibrous tissue, like most abnormal fibrous tissue, has begun to contract; therefore the surface of the liver is uneven, and if the prominences of liver substance between the strands of contracting fibrous tissue are of a suitable size the liver is hobnailed. Many of the liver-cells are atrophied, and many have undergone fatty degeneration. These are the only essentials of a cirrhotic liver. Its weight is very variable, and may be anything between thirty and two hundred ounces, according as to whether the atrophy of the liver-cells, the fibrous tissue, or the fat preponderate. The liver may be jaundiced, and on section the fatty degenerated portions of the liver substance may stand out raised from the surface of the section, because cutting the organ has relieved them from the pressure of circumjacent fibrous tissue. The capsule of the liver may be a little opaque, and occasionally the portal vein is thrombosed. I particularly want you to remember that a cirrhotic liver is easily recognized without the aid of a microscope.

I have noticed that students find cirrhosis of the liver a difficult subject to understand, but the difficulties are entirely due to the fact that many authors have described cirrhosis incorrectly and that other authors have copied these incorrect descriptions. If you simply observe for yourselves the cases that occur in the wards and the post-mortem room, I think your difficulties will disappear. The first difficulty that you usually encounter in reading about cirrhosis is that you are told there are two varieties of cirrhotic liver,—namely, the atrophic and the hypertrophic,—and that each variety has its own special symptoms, so that atrophic cirrhosis and hypertrophic cirrhosis are separate diseases. This distinction we owe to French authors. I shall try to show you that if we observe for ourselves we shall find that no such distinction exists.

The French teach that there are the following points of distinction between atrophic and hypertrophic cirrhosis: Firstly, the large or hypertrophic cirrhotic liver is not hobnailed; the increase of fibrous tissue starts round the biliary channels, and this new tissue surrounds individual lobules and extends into them; for these reasons it is called biliary cirrhosis, and is said to be perilobular and intralobular; there is, too, a plentiful formation of new bile-ducts.

On the other hand, they say that the small atrophic liver is hobnailed; that the formation of new fibrous tissue starts round the portal vein; that it embraces several lobules in one area and never extends into the lobules; for these reasons it is called portal cirrhosis, and is said to be multilobular and extralobular; there is no new formation of bile-ducts. With regard to symptoms, the French state that when the patient's liver is affected with the hypertrophic form of cirrhosis he will have little or no ascites; his jaundice will be marked and lasting; he will be particularly liable to delirium, convulsions, and coma; the spleen will be found to be enlarged; he will have occasional attacks of slight pyrexia; there will be no albumen in his urine, and he will from time to time complain of attacks of hepatic pain. Further, they state that sufferers from hypertrophic cirrhosis are not addicted to alcohol, that they are younger than those who suffer from atrophic cirrhosis, that they are mostly men, and that their illness lasts a long time. On the other hand, the sufferers from atrophic cirrhosis usually have considerable ascites; jaundice, if present, is slight and often transitory; the spleen is not enlarged; delirium, convulsions and coma, slight pyrexia, and pain are not so often met with as in the hypertrophic disease, and albuminuria is not uncommon. Also, they have usually taken excessive amounts of alcohol, they are older than those who suffer from the hypertrophic form, most of them are women, and their illness lasts a shorter time.

Now, I quite admit that if you see many cases of cirrhosis of the liver you will sometimes meet with those in which the liver and spleen are enlarged and the patient is jaundiced but has no ascites. But cirrhosis of the liver is a very common disease, so that when you have seen many cases you will meet with all sorts of groupings of the symptoms; this, however, is no justification for establishing different varieties of the disease, any more than you would be justified in establishing as a distinct variety of typhoid fever those cases which had abundant diarrhoea and no spots, although by the time you have seen many cases of typhoid you are sure to come across several such. However, let us take separately each of the points urged by the French school and see if they are valid.

First, as regards the appearance and weight of the liver. You may often see a hypertrophic liver which is hobnailed. The first patient whom I described to you to-day had a liver which weighed

seventy-four ounces; that is to say, it was considerably hypertrophied, but yet it was markedly hobnailed. Of course, as a rule, the smaller the liver the more marked is the hobnailed appearance, for contraction of the fibrous tissue means that the liver will become small and that the hobnails will become well marked; this very simple consideration should at once prevent any one from exalting into a position of great importance the fact that atrophic livers are markedly hobnailed. Let us now examine the weight of some cirrhotic livers: The cirrhotic liver may weigh anything between thirty and two hundred ounces. Dr. J. A. P. Price published an account of one hundred and forty-two fatal cases in the Guy's Hospital Reports. In thirty-three the liver weighed between fifty and sixty ounces, in twenty-nine it was under fifty ounces, and in eighty it was over sixty ounces. So you see that, as the weight may be anything, it is impossible to group the livers into a hypertrophic group and an atrophic group, for, if you do, where will you place the thirty-three in which the weight was between fifty and sixty ounces? Further, as more of the cases were over than under the normal weight, they strongly suggest that the course of events in cirrhosis is for the liver first to become large and then to become small, for then, as the patients die off, the number would diminish, and we should have fewer with the light livers, and some—viz., those in whom it weighed between fifty and sixty ounces—would die when the liver, having been hypertrophied, had shrunk down again to a normal weight. This view is certainly correct for some (and probably all) cases, for Dr. F. Taylor has, in the Guy's Hospital Reports, published a case in which a big liver was known to shrink while the patient was under observation, and other authors have published similar cases.

Now let us turn to the next point on which the French lay such stress,—namely, that when the liver is large, ascites, if present, is slight. Our very first case disproves this contention, for the liver weighed seventy-four ounces, and yet within three weeks the patient was tapped twice and thirty-six pints of fluid were withdrawn. I could quote you many other similar cases.

Then, too, our first case shows it is not true that when the liver is enlarged the jaundice is deep and long lasting, for the jaundice in this patient was very slight, and certainly my experience is that there is no relationship between the jaundice and the size of the liver.

Nor have I ever been able to notice any relationship between the size of the liver and the nervous symptoms. The liver in our second case is certainly not enlarged, and yet the patient is drowsy, which is contrary to the French view.

You can easily test the statement that when the liver is enlarged the spleen is enlarged, and I can promise you that you will find there is no relationship between the enlargement of the liver and that of the spleen. The most recent collection of cases is Rolleston's, and his figures show most conclusively that there is no such relationship.

The next point we took among those urged by the French was their statement that when the liver is enlarged the patient does not give an alcoholic history. This is altogether contrary to my experience, and our first patient had a hypertrophied liver, and yet she had drunk hard. Whether the liver is large or whether it is small, you are equally likely to find that the patient has been intemperate, and, indeed, in the collection of cases made by Rolleston, a history of alcoholic intemperance was rather more often found in those who had large livers than in those who had small.

I have never observed albuminuria to be commoner when the liver is small than when it is large, and some series of cases show granular kidneys to be met with oftener in association with a large liver, while other series show they are more often associated with small livers; so we see that this supposed difference between hypertrophic and atrophic cirrhosis does not hold.

The French do not lay much stress on the point that the hypertrophic cases are the most likely to have attacks of pain and slight pyrexia, and I have never noticed any differences in these respects.

Their statement that the atrophic cases occur in older people than the hypertrophic is, I think, true, but the distinction is of no value, for as the atrophic stage succeeds the hypertrophic, it naturally follows that, as a rule, the sufferers from atrophic cirrhosis will be older than those who have hypertrophic cirrhosis.

I took fifty cases at random to see whether it is true that the atrophic form is common in women, and there was no noteworthy association between the sex and the size of the liver; indeed, in this series the hypertrophied liver occurred slightly oftener in women than men.

The statement that the hypertrophic form lasts longer than the

atrophic is of no value whatever as a distinction, as it is obvious that it must be so, for many of the hypertrophic cases will live on through this stage, but when a man gets to the atrophic stage he has no further to go, and is near his end.

There has been much controversy both here and abroad as to whether the French statements as to the histological distinction between the two holds, and it has been conclusively shown that there is no such distinction. I think you will now agree with me that there is no distinction between atrophic and hypertrophic cirrhosis, and so one of your greatest difficulties in understanding the subject is swept away.

The next difficulty to which I want to direct your attention is that of biliary cirrhosis. Charcot and others have tied the common duct in animals, and they say that by this means they have produced cirrhosis of the liver; further, that it was a cirrhosis having all the characters of what the French school call hypertrophic cirrhosis. I have already given you the characteristics which the French assign to what they call hypertrophic cirrhosis. Charcot and his followers further maintained that when in the human subject the duct is obstructed for some time, as it often is by a gall-stone, the liver becomes affected with hypertrophic cirrhosis. I myself have never seen any evidence that gall-stones can produce cirrhosis, and the question has been recently definitely settled by Naumyn in his important book on gall-stones. He says that the great majority of sufferers from biliary obstruction show no evidence of cirrhosis, and if in a large series of cases one or two show a little cirrhosis, it is very slight and often requires a microscope for its demonstration; it is never marked enough to cause any symptoms, and has not the characters which the French describe under the term of hypertrophic cirrhosis. So you may dismiss biliary cirrhosis from your minds, for, clinically, it does not exist.

Another difficulty is cirrhosis as it affects children. In the first place, you must remember that it is decidedly rare, and that at least three varieties exist. The first and commonest is a cirrhosis which in its clinical symptoms exactly resembles ordinary cirrhosis of adults. Very often children who suffer from this have taken alcohol, and you must not be too ready to assume that they have not, for, naturally, their parents deny having given them any; still, in a very



few cases it appears that the children have certainly not taken alcohol, and in these cases we do not know the cause of the cirrhosis.

The second variety is very rare. It was first described by the French, especially M. Hanot. The liver is very large, the spleen reaches below the umbilicus, the child is deeply jaundiced, and the fingers and toes become enlarged. We understand nothing about this disease, but it is so rare that most practitioners never see a case. However, you have all, I hope, seen the boy under Dr. Taylor's care in Stephen Ward who is suffering from it.

You will find described by Dr. Taylor in the Guy's Hospital Reports, Vol. 52, the very exceptional case in which, in a child, the disease lasted a very long time, although many of the ordinary symptoms of cirrhosis were absent and no cause could be made out for the disease. This probably represents an extremely rare variety of children's cirrhosis of which we know nothing.

Another difficulty about which there is much confusion is the etiology of cirrhosis. The following causes have been given for cirrhosis:

*Alcohol.*—In over sixty per cent. of hospital cases you will easily obtain a history of excess in taking alcoholic drinks. When you remember that persons who drink often try to conceal the fact, and that in hospital patients the history of previous habits is often very difficult to obtain, it will be clear to you that over-indulgence in alcoholic drinks is by far the commonest cause for cirrhosis; indeed, it is possible it may be the only cause. You will notice I have ascribed cirrhosis to alcoholic drinks and not to alcohol, for we do not know what constituent of the alcoholic drinks causes cirrhosis; that it is not ethyl alcohol is quite possible, for it is impossible to produce cirrhosis in animals by the administration of absolute alcohol, and cirrhosis is much less common in Scotland than England. These facts suggest that cirrhosis may be due to some of the various other alcohols, or to the oils that are present in alcoholic drinks, for, as they are not equally present in all, some alcoholic drinks may produce cirrhosis, while others may not.

*Syphilis.*—I particularly want you to be clear about this. Acquired syphilis produces gummata in the liver, which, when absorbed, leave large depressions on the surface of the organ. It also causes large fibrous bands to appear in the liver, and as these contract they, too, deform the organ; further, here, as in any other

part of the body, when gummata are absorbed they leave much fibrous scar tissue. You will see, therefore, that, although the surface of the syphilitic liver is irregular and although the fibrous tissue in it is increased, these irregularities and the fibrous tissue are not uniform, and the liver never resembles an ordinary cirrhotic liver. The two diseases are so distinct that any student who could not tell a cirrhotic from a syphilitic liver ought at any examination in medicine to fail. Congenital syphilis produces the same effects as acquired syphilis, and they can, during life, be made out in childhood, but if the sufferers from it survive, the liver so far recovers itself that it cannot during life be recognized as deformed by the time adult age is reached. Congenital syphilis may also increase the fibrous tissue in the liver to such an extent that the organ becomes enlarged, stony hard, inelastic, flint gray, and anæmic. The sufferers from this form of disease of the liver are nearly always still-born, or, at any rate, they only live a few days.

Many books tell you that syphilis will produce cirrhosis because it causes an excess of hepatic fibrous tissue, but I think I have made it clear to you that it does not produce effects which in any way resemble ordinary cirrhosis of the liver, and therefore it is much better not to speak of syphilis as a cause of cirrhosis, especially as a syphilitic liver never produces the symptoms of ordinary cirrhosis of the liver.

*Malaria.*—Repeated attacks undoubtedly often leave the liver very hard and of considerable size, but whether malaria ever produces a liver like the common cirrhotic liver, in its morbid anatomy and symptoms like the ordinary symptoms of cirrhosis, is doubtful. I have only seen one case in which this might have been so, and in this, as in so many of these cases, it was very difficult to be sure that alcohol might not have been the cause. If malaria is a cause for cirrhosis, it is very rare.

*Backward Venous Pressure from Cardiac or Pulmonary Disease.*—Long-standing venous congestion of any organ leads to a slight increase of the fibrous tissue in it, but venous congestion of the liver never leads to anything like enough new fibrous tissue for it to be possible to confound such a liver with an ordinary cirrhotic liver, nor are the symptoms of cirrhosis associated with the condition (nutmegged) of liver met with in the subjects of cardiac and pulmonary disease.

*Lead-Poisoning, Tuberculosis, Dyspepsia, Diabetes, Pneumonia, Measles, and Scarlet Fever.*—Some observers have maintained that in each of these diseases there is occasionally an increase of fibrous tissue. Even if this is true, the increased fibrous tissue is minute in quantity, the microscope always being required for its detection, and neither clinically nor after death does the liver ever resemble ordinary common cirrhosis of the liver.

Therefore, we learn that for the disease which has its own symptoms and its own typical morbid anatomy we only know one cause,—namely, alcoholic drinks. This is certainly by far the commonest cause, but it may be that some few cases own some other cause or causes, although, if so, we do not know them; certainly, none of the commonly mentioned causes (except, perhaps, in rare instances, malaria) ever produce the disease called cirrhosis of the liver.

We may, therefore, sum up as follows: Some substance in certain alcoholic drinks acts as a chronic irritant to the liver and produces the changes in it that we have already described. The liver enlarges and subsequently shrinks, the shrinkage being due to atrophy of liver-cells, contraction of fibrous tissue, and absorption of fat. This view, that the large liver is the early stage and the contracted liver is a later stage of the same disease, satisfactorily explains the following facts: (1) The large liver has been observed to contract; (2) the large liver is more commonly seen in the wards than the small, because many sufferers from cirrhosis die before the liver becomes small; (3) the age at death of those with large livers is some four years less than that of those with small; (4) at death a large liver is oftener found than a small, and the liver is often of normal weight.

Now, I wish to point out to you a few facts about the symptoms. In the first place, it is important to remember that the liver may be cirrhotic and yet the patient may have no symptoms of cirrhosis, so that until the physician, while making a routine examination of his patient, finds the liver enlarged and rough, both he and the patient are unaware that it is diseased. Thus, you may find it accidentally in patients whom you examine for life insurance. In between a third and a half of all the cases of cirrhosis found in the post-mortem room the patient has died of something else than cirrhosis, and in many of these cases, although he has been under observation in the wards, no symptoms have been observed pointing to cirrhosis. Cir-

rhosis of the liver is frequently found in those dying of tubercle, chronic Bright's disease, and pneumonia because alcoholic excess is particularly fatal to sufferers from these diseases, but it may be found in those who are apparently perfectly healthy and are killed by some accident. Those persons who, when they come under observation, have a cirrhotic liver without any symptoms of cirrhosis may, if they give up drink, continue to live for many years without showing any symptoms.

But a patient with a cirrhotic liver may at any time develop the symptoms of the disease, and if he continues to drink and lives long enough he will certainly do so. These symptoms, which have been illustrated by our two cases, are:

(1) Sallow anæmia, with a tendency to epistaxis and to the development of purpuric spots; (2) a red nose, often going on to acne rosacea, together with injected venules on the cheeks; (3) morning vomiting, loss of appetite, indigestion, and constipation; (4) piles, hæmatemesis, and bleeding from the gastro-intestinal mucous membrane; (5) jaundice; (6) scanty, high-colored urine, which contains an excess of urates but little urea; (7) ascites; (8) œdema of the feet; (9) coma—or more rarely delirium with convulsions—and difficulty of breathing.

Some of these symptoms, as vomiting, indigestion, and constipation, may be due to catarrh of the stomach, and, as the anastomoses between the gastric and œsophageal veins are often found dilated, the cirrhosis may, by its impediment to the portal circulation, cause hæmatemesis, piles, melæna, and perhaps enlargement of the spleen. The jaundice may be a direct result of disease of the liver, but the ascites, the swelling of the feet, and the nervous symptoms require special consideration. The ascites is not solely, if at all, caused by pressure on the portal vein, for I have collected a number of cases which show that the fluid collects very rapidly, often at the rate of from a pint to a pint and a half a day, and you will notice how rapidly it collected between the two tapings in our first case; further, the onset of ascites nearly always means that the patient will be dead within two or three months of its onset, so you see that in a chronic disease we usually have a rapid outpouring of fluid into the peritoneal cavity nearly always associated with a fatal issue. Therefore, the sole cause for ascites cannot be pressure on the portal vein, for in cirrhosis it is slow and gradual and would not lead to a sudden

outpouring of fluid into the peritoneal cavity; further, you must remember that it is difficult in the lower animals to produce ascites by ligation of the portal vein.

The swelling of the feet is not due to pressure of the fluid in the abdomen on the vena cava, for I have seen many cases (like our second case) which show that the œdema of the feet often precedes the ascites, and, too, it bears no relation to the size of the liver. These facts all point to the view that the patient suffering from cirrhosis of the liver is like the sufferer from chronic Bright's disease in that he is always liable to the somewhat sudden development of a series of symptoms, usually fatal, and they also suggest that, as with the uræmic symptoms of Bright's disease, so these symptoms are due to the manufacture by the diseased organ of, or its failure to excrete, some toxic substance, and that this substance is a coma-producing lymphagogue (it causes ascites and œdema of the feet) which nearly always kills.

We have no time left to discuss the treatment. Our first patient had copaiba resin because Dr. Taylor has pointed out that this is an excellent diuretic for cirrhotic patients suffering from ascites, and I can certainly confirm what he says.

# Gynæcology and Obstetrics.

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## THE TREATMENT OF OVARIAN CYSTS COMPLICATING PREGNANCY AND LABOR.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL.

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GENTLEMEN,—An ovarian cyst as a complication of labor is a rare event, occurring but once in some three thousand five hundred births in the Berlin Maternity Clinic and with much less frequency in general practice. It is not strange, therefore, that the best treatment of this complication is as yet unsettled. The experience of any one individual is so small that few indeed are entitled to speak on the matter with any authority.

The present idea of the treatment of ovarian cysts complicating pregnancy and labor entertained by physicians in general may, I think, be expressed as follows:

If the tumor is discovered during the first four or five months of pregnancy its removal should be attempted. Ovariectomy during gestation is not necessarily difficult or dangerous, nor does it, as a rule, interrupt pregnancy. From statistics of one hundred and thirty-five operations it appears that the mortality was less than six per cent., and the pregnancy was interrupted by the operation in about twenty per cent. of the cases. With this opinion I am in entire accord, having had a gratifying personal experience with the operation in pregnancy. If the tumor is first discovered after the fifth month of pregnancy or after the woman has fallen in labor, if it has become displaced downward into the pelvic cavity, and is incarcerated there behind the womb and under the promontory, resisting all efforts to displace it upward, then a puncture through the vaginal

vault, preceded by a thorough cleansing of the vaginal mucous membrane and with a thoroughly aseptic technique, is said to give the best results in labor. This idea, prevalent among physicians in general at present, is, in my opinion at least, incorrect. While it may be safer for the general practitioner without special surgical experience to puncture an ovarian cyst through the vaginal vault, it is vastly better for a competent surgeon to perform a Cæsarean section and at the same time to remove the tumor, taking with it in certain cases the womb, but, if possible, leaving the uterus behind, as the obstruction to labor can be permanently removed, and there will be no obstacle in the pelvis to obstruct future deliveries.

The first case of the kind which I encountered in practice was treated by the method at present recommended routinely in almost all text-books on obstetrics,—namely, by puncturing the cyst through the vaginal wall. The bulk of the tumor could not be satisfactorily reduced, as there was a large amount of solid matter in it and it was impossible to tap it in all its loculi. Version was then performed, and the child extracted with so much difficulty and by the use of such great force that its neck was fatally injured. The tumor was so bruised by the passage of the child through the pelvis that it became gangrenous, and it was necessary to operate on the woman, who had fallen into a truly desperate condition, during her puerperal convalescence. Fortunately she happened to recover, but her escape was a very narrow one.

Since then I have performed Cæsarean section in two other cases of ovarian cysts impacted in the pelvis with a perfect result for both mother and child. In the first case the woman had been in labor four days, and the lower uterine segment and the pelvic connective tissue were extraordinarily œdematous. The endometrium of the womb was almost black in color, so that it appeared to be necessary to do a hysterectomy. The patient made an afebrile convalescence, and the child was delivered alive and is living and well to-day, more than a year after the operation.

The second patient upon whom you saw me perform a Cæsarean section and who is now exhibited with a perfectly healed wound was operated on at a selected time some ten days before the date for labor. The child was delivered in the usual manner through the uterine incision, and the tumor, a large dermoid cyst, was then excised. As the pelvic organs were in excellent condition, the

wound in the uterus was sewed up by interrupted and continuous catgut sutures with a separate peritoneal stitch, and the woman made a good recovery. The child was born alive, and is thriving at the present time.

Any one who has had experience with the two methods of treating these cases should have no hesitation as to the choice between them. The Cæsarean section is perfectly certain, is in accord with modern surgical principles, relieves the woman immediately of her tumor, and practically insures her an uncomplicated and safe convalescence in her puerperium. Even if the cyst could be completely evacuated by vaginal puncture there is a constant danger hanging over the woman of gangrene of the tumor during the puerperium, and at the very best an abdominal section must be performed some time later for the removal of the cyst.

The difficulty in the diagnosis of these cases also makes vaginal puncture an extremely uncertain procedure. It is quite impossible to tell with absolute certainty whether an ovarian tumor is a dermoid cyst or a multilocular cyst. In the writer's last case of an ovarian tumor, which was as large as a foetal head, two distinct loculi were present. In one there was more than a pint of the ordinary ovarian cyst fluid, while in the other and larger one there was the usual contents of a dermoid cyst, consisting of sebaceous matter and hair. Had this tumor been punctured and had the dermoid contents entered the peritoneal cavity, which would indubitably have happened through the puncture-hole, the woman unquestionably would have lost her life from septic peritonitis. There is no such putrescible matter as this dermoid cyst contents, and nothing which so rapidly undergoes putrefaction in the peritoneal cavity, with such sure production of an uncontrollable profuse suppurative peritonitis. In this case a clear and distinct differential diagnosis between dermoid cyst and multilocular dermoid cyst would have been impossible, as in the two portions of the cyst were felt the characteristics of each kind of growth. As a matter of fact, as already stated, it was found after removal by operation to be a combined tumor.

It is quite impossible also to make a distinct diagnosis in every case between a fibroid tumor and an ovarian cyst impacted in the pelvis. If a trocar were plunged into a fibroid growth the bulk of the tumor would of course not diminish, while the woman would run a considerable risk of a fatal hemorrhage into the peritoneal cavity.



The uncertainty of the result, therefore, together with the doubt as to the sufficient reduction in the bulk of the tumor, and the possibility of subsequent gangrene of the tumor and infection of the peritoneal cavity, make vaginal puncture an operation of such risk that the modern specialist at least should never again consider it.

Statistics of two hundred and seventy-one cases, collected by Heiberg, show a maternal mortality of about fifty per cent. in ovarian tumors obstructing labor whether the child was delivered by forceps without puncture, by version without puncture, or by either of these operations following puncture of the cyst. There is no question but that a vastly better record than this can be obtained by the modern abdominal surgeon with Cæsarean section and coincident removal of the tumor.

## A CASE OF LABOR IN A UNIVERSALLY CONTRACTED PELVIS, WITH A PROMONTORY MARK ON THE HEAD.

CLINICAL LECTURE DELIVERED AT THE ROYAL MATERNITY HOSPITAL.

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GENTLEMEN,—At our last clinic I showed you a patient in whom labor was beginning. We noted an unusual prominence of the abdomen, which was found to be due to the fact that the head could not lie within the pelvis. The patient was a primipara in whom at full time we should expect to find the head in part below the brim; in this case it was riding above it. That this was due to the lower uterine segment not being able to sink into the pelvis was evident also from the unusually high position of the bladder. With only fifteen ounces of urine, we found it extending five inches above the pubes, and when emptied it was still seen to be partly an abdominal organ. This high position of the bladder could not be the result of labor, the first stage of which had just begun, but must have been part of the general displacement of the soft parts, due to pelvic contraction.

The measurements of the pelvis were as follows: interspinous diameter, nine and one-quarter inches; intercrystal diameter, nine and one-quarter inches; external conjugate, seven inches, diagonal conjugate, four and one-quarter inches. From these we inferred that the true conjugate was about three and one-half inches.

There are two types of pelvic deformity which you will meet with most frequently in practice. In the first one, all the diameters are reduced in proportion, the result of which is a pelvis whose shape is normal but whose size is small. Strictly speaking, it is not deformed,—that is to say, there is no departure from the normal form. The second is truly deformed, the pelvis being flattened from before

backward. The chief result of this flattening is seen in the brim, in which the normal heart shape is flattened out into a kidney. The form of the false pelvis is also affected in extreme cases, the result of the flattening here being to throw apart the anterior spines and thus diminish the curvature of the crests. Both the absolute and relative lengths of the transverse diameters of the false pelvis thus come to be of significance.

To which of these types does this pelvis conform? The measurement of the false pelvis in this case brings out two points,—that the pelvis as a whole is narrowed across and that the curvature of the iliac crests is diminished. The external conjugate shows that the true conjugate must be reduced by not less than half an inch, while the length of the diagonal conjugate gives the reduction as something between one-quarter and three-quarters of an inch, according as we suppose the pelvis to be of the equally contracted or flattened type.<sup>1</sup>

We have estimated the length of the true conjugate as three and a half inches.

Another point noted on examining this patient was that the transverse diameter of the brim was shorter than usual. We have no means of gauging the length of this diameter accurately; we have a hint of narrowing in the absolute shortening of the false pelvis transversely. The best mode of estimating it is with the hand placed internally, so as to judge of the space relative to the foetal head. In the case before us we found that the transverse diameter was diminished, as well as the conjugate. In addition to this feature of general contraction there was an unusual projection of the promontory, as we shall see when we come to look at the head of the child after birth. This pelvis, therefore, combined both the common types of pelvic deformity. The brim was not only reduced in size all round, but the notch in the heart was deeper than usual through the projection of the promontory. This latter feature makes it approximate to the kidney-shaped type of brim.

In considering what was the best thing to do for our patient, we set on one side Cæsarean section, as the contraction was not sufficient for that operation. With this degree of contraction we may

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<sup>1</sup> The difference between the diagonal and the true conjugate is, in a pelvis of normal build, one-half an inch; in the flat, rickety pelvis this is increased to one inch on account of the greater depth and eversion of the symphysis.

still hope to get a living child *per vaginam*. We have to consider the alternative measures of delivery by forceps or by turning. To understand the principles which guide us in choosing the method, we must consider how a change in the size or shape of the pelvis affects the mechanism of labor, and for this reason, that the mode of treatment is best which most closely follows nature. We take our clue as to the management from the natural mechanism. The mechanism of labor is simply a question of accommodation, depending on the one hand on the shape and size of the pelvis, and on the other of that of the foetal head.

Where the form of the pelvis remains normal, the mechanism is normal, any reduction in size simply exaggerating the normal accommodation movements. So also the moulding of the head produced by labor is normal in character though extreme in degree. In the flattened pelvis, on the other hand, the form is altered, the heart being changed into a kidney by the encroachment of the promontory on the space of the brim. This change in form produces a change in mechanism. To begin with, the head lies differently at the brim. It is transverse, extended, and canted over, instead of being oblique, flexed, and with the sagittal suture as its lowest point. Further, it passes the brim extended instead of flexed. It would take us too much aside to dwell on these details; it is sufficient to know that the head passes the brim by extension, that it is prepared for this by lying extended at the brim, and that this departure from the usual mechanism is related to the departure from the normal form through the projection of the promontory.

Comparing now delivery by forceps and by turning, as regards the effect of the position of the head in passing the brim, we note that forceps causes flexion of the head, while turning produces extension. From the size and shape of this pelvis, and especially from the narrowing in the transverse diameter, we might infer *a priori* that it is impossible for the head to pass this brim extended; and the form of the head after delivery, in this case, which we shall refer to later, confirms this. But the reasons why we dismiss turning lie in the shape of the pelvis and its effect on the mechanism of labor. While this pelvis is contracted in the conjugate, there is a corresponding contraction in the transverse, making the brim normal in shape though abnormal in size. The head will therefore pass by the normal mechanism of flexion, and to deliver by turning would destroy

this mechanism. Were you to turn in this pelvis, the head would extend and become jammed at the brim. Another reason against turning is that the patient is a primipara, in whom you will find the necessary manipulation difficult from the rigidity of the soft parts. For this case, then, forceps is the only mode of delivery.

To facilitate the passage of the head in a case of this nature the operation of symphyseotomy has been done of recent years. The division of the symphysis allows separation of the pubic bones to a certain extent, by which more room is gained. While extensively practised in France and Italy, it has not found much acceptance in this country, as the amount of space gained is not of great practical importance.

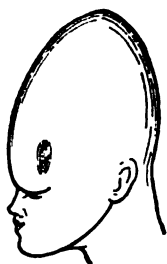
When we saw this patient on the Wednesday afternoon, the first stage had already begun, the cervix being dilated to the size of a five-shilling piece. The membranes did not rupture till Thursday at noon. The first stage is usually long in cases of deformed pelvis, probably because the uterine contents cannot descend so well into the lower uterine segment. Under these circumstances there is nothing to be done except to wait; any interference is undesirable, and especially anything that would lead to premature rupture of the membranes. This is an accident which is specially liable to occur, in cases of labor with deformed pelvis, just when it is most undesirable. You can understand the importance of having the cervix fully dilated by the natural methods as a preliminary to operative interference in delivery.

The patient was delivered with axis-traction forceps. A considerable amount of force was necessary, as is evident from the marks of forceps on the child's head. Unfortunately, the child was still-born. The heart continued beating for over half an hour; but, though artificial respiration by Schultze's method was kept up during that time, natural respiration could not be started, owing probably to some injury to the respiratory centres.

On looking at the foetal head (Fig. 1) you will notice the peculiar shape, and especially the dent on the left frontal bone immediately above the left orbit. The head is elongated in the occipito-mental diameter, due to compression in the suboccipito-bregmatic circumference. This produces a sugar-loaf head, and the form is quite characteristic of an equally contracted pelvis. If you see a head like this you can be absolutely sure that the pelvis was of that

form. The head is in fact a cast of the pelvis. The moulding is the same as you meet with in ordinary cases of labor, only it is extreme. Just as we saw that the pelvis is normal in form, but extremely small, and the mechanism of labor normal in character, but exaggerated, so the moulding is also normal in character, but pronounced. This dint on the left side of the forehead is, however, unusual; from its shape it must have been produced by a bony prominence; in fact, the only thing that could produce it is the promontory of the sacrum. It has this interest that it enables us to place the head in the exact position in which it passed the brim. As it is over the left orbit, the head must have passed with the occiput to the front and

FIG. 1.



Head delivered through justo-minor pelvis by forceps; promontory mark above the left orbit.

FIG. 2.



Brow presentation, with promontory mark above the right ear.

left side, lying between the right oblique and conjugate diameters. This is almost the normal position, except that a certain amount of rotation occurred as the head was pulled through the brim. In a normal pelvis this rotation does not, as a rule, occur, until the head is at a lower level,—that is, until the head has reached the floor of the pelvis.

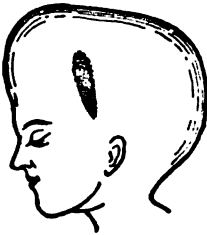
This promontory mark proves that the pelvis was not only equally contracted, but had a slight projection of the promontory,—that is to say, was also slightly flattened. This flattening, however, was not sufficient to disturb the mechanism of labor, the head passing the brim, as already said, in flexion.

The winter before last we had a very interesting case of labor in a pelvis similar to this, only that in it the projection of the promontory was more marked than the general contraction. Here you have a drawing of the foetal head. (Fig. 2.) From the shape of it you can tell that it was a brow presentation. The promontory mark occupies quite a different position from what it does in the case

before us: it is an oblique groove above the ear. From the appearance of this head we infer that the projection of the promontory was so marked that it disturbed the natural mechanism and caused extension instead of flexion; hence the brow presentation. Further, the situation of the promontory mark shows that the head passed the brim lying in the transverse diameter of the pelvis, and with the face leading.

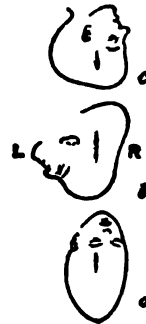
This third head (Fig. 3) shows the usual position of the promon-

FIG. 3.



Vertex presentation from flat, rickety pelvis, with promontory mark in front of the left ear.

FIG. 4.



Showing how the presentation can be recognized afterwards from the position and direction of the promontory mark.

tory mark in the case of the flat, rickety pelvis. We shall discuss its mode of production when we have a case of labor in a typical flattened pelvis. In the mean time will you note its position and character for the sake of comparison with these other two heads? It is a vertical furrow in the front of the ear. (Fig. 3.) From its direction we infer that the head passed the brim with the vertex leading.

A case like this shows us the value of looking carefully at the child's head after birth. Its shape and the marks upon it give us important corroborative evidence as to the character of the pelvis. To bring out this point, let us place these three heads below each other and in such a position that the promontory furrow in each is in line. They are now lying (see Fig. 4) as they passed through the pelvis. The letters *L R* indicate the left and right sides of the mother's pelvis. As the promontory mark faces you, it is as if you were looking at the pelvis from behind. And now you see that *a* has been a vertex presentation left occipito-anterior, while *b* has been a brow, and *c* a vertex with the head transverse and extended.

## PYOSALPINX, WITH A STUDY OF FOUR CASES.

CLINICAL LECTURE DELIVERED AT THE LOUISVILLE CITY HOSPITAL.

BY LOUIS FRANK, M.D.,

Professor of Bacteriology in the Kentucky School of Medicine; Gynæcologist to the Louisville City Hospital, etc., Louisville, Kentucky.

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GENTLEMEN,—Two of the cases which I shall bring before you to-day, you will remember, were operated upon some time ago, and I will briefly outline the history and the treatment that were carried out. In conjunction with these we will operate upon two other cases, and discuss them as fully as our time will permit.

CASE I.—The first patient, aged eighteen years, was operated upon three weeks ago, and, as you see, she has made a perfect recovery, being now ready to go home. We bring her before you not only to discuss the different aspects of the case, but also to show you the result of the operation. The specimens, which you will remember, were exhibited at the time of their removal, consist of a pyosalpinx from one and a tubo-ovarian abscess from the other side. The tumors are typical, both of them containing pus. The patient is married and gave the history of a miscarriage, followed by fever, swollen belly, odorous discharge, and a rather protracted convalescence, so that I think we may look upon the disease as of puerperal origin.

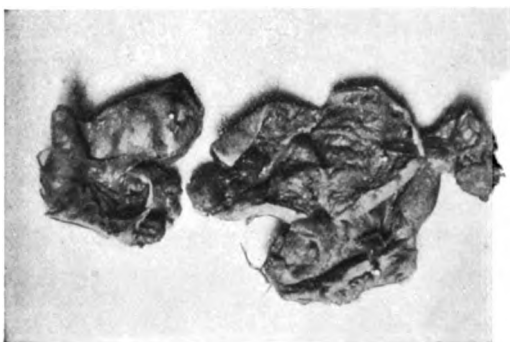
Puerperal fever, or childbed infection, is, as I have frequently told you, one of the most frequent factors in the production of tubal troubles, it being accountable for a very large percentage of all cases of this nature. Not always, however, do we have pyosalpinx or ovarian abscess together or alone resulting from puerperal infection. We may have, as has been demonstrated to you, an abscess of the ovary without infection of the tube; or we may have pus in the tubes, the ovaries remaining normal so far as any pathological condition within the investing tunic is concerned. Again, we may have a puerperal infection which will run its course without any involve-



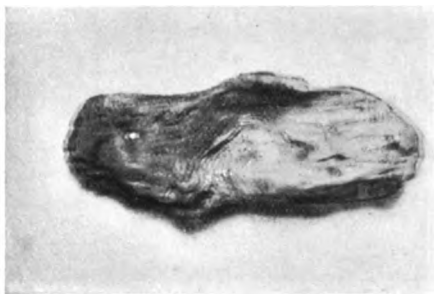
ment of these structures whatsoever; the trouble may become localized in the broad ligaments, resulting in the formation of local foci of pus which may break down and discharge into the vagina, rectum, bladder, or uterus, or which may run a chronic course, and by gradually enlarging give rise to much trouble, or which may later invade the peritoneum and set up a fatal peritonitis. Again, we may have the poison confined to the uterus, the patient suffering from a toxic infection or toxæmia rather than a true septicæmia. There may be small abscesses in the walls of the uterus, or the organisms may gradually invade the uterine muscular coat. If the latter penetrate deeply enough, they will find their way into the peritoneum and set up a peritonitis with extensive adhesions, often involving the tubes or ovaries, with the production of pus secondarily, matting the structures together. Such conditions result in some of the worst cases of pelvic troubles with which we have to deal. These pelvic diseases, inflammatory in character, due to infection with the various pyogenic organisms which cause this trouble, are among the most difficult cases for successful operation that we have. The adhesions, as you have often been told, are exceedingly dense, the pus is, as a rule, of the most virulent character, making these cases extremely trying ones. Great caution must be exercised in enucleating the masses to prevent peritoneal infection. Should you have to deal with pus-tubes, with or without ovarian abscesses, bound down by dense adhesions, they may often be evacuated through the vagina, the pus being drained, and later secondary operations for removing the diseased structures performed. However, we will not now enter into a discussion of the relative merits of vaginal and abdominal operations, nor the indications for one or the other procedure.

This case, as you will remember, was operated upon suprapubically, and the result speaks for itself. In no method of procedure could the result have been better. The patient will tell you that she is absolutely free from pain and suffers no inconvenience whatsoever.

CASE II.—Let us now consider the specimens from the other patient, operated upon at the same time, and contrast the difficulties in operating and also the condition present in the abdomen, bearing in mind the etiology of each. The patient is twenty-nine years of age, married, the mother of four children, the oldest of which is nine years and the youngest two and one-half years of age. The trouble



**FIG. 1.—CASE I.**—Double pyosalpinx with tubo-ovarian abscess, puerperal in origin.



**FIG. 2.—CASE II.**—Right pyosalpinx, gonorrheal in origin.



is one of gonorrhœal origin, as the history will clearly show. She told us that she had had four children, no trouble following the birth of any of them, in fact, her menstrual and puerperal history is as clean as we could desire. We found further, upon questioning, that she had been separated from her husband, and inquiry into the cause develops the fact that she was infected by him, as she says, with gonorrhœa. Following this gonorrhœa, at exactly what time she does not now remember, she began having some menstrual trouble, the pain becoming of such a severe character that she could hardly bear it. She had been confined to her bed off and on, had been losing flesh, and, in fact, going down hill for the past few months.

An examination at the time of her admittance, as you will remember, revealed a mass in one side of the pelvis only. The uterine infection seemed to have run its course. There was not much discharge from the vagina, and in the hope of being able to remove the adnexa of only one side we decided to operate from above. The uterine cavity was first curetted, mopped out with carbolic acid and then packed with aseptic iodoform gauze. The abdomen having been prepared a median incision was made, and while we found the uterus retroflexed and fixed, with adhesions upon both sides, the disease had not, we thought, impaired the utility of the tube and ovary upon the left side. After freeing all adhesions a large pyosalpinx was brought up, ligated, and removed. The broad ligament was very much thickened, so that rather a large pedicle was necessarily left.

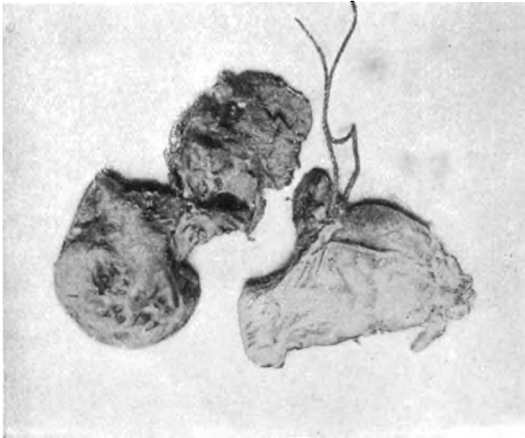
Now to compare this case with the one previously spoken of. You will remember how much more easily these adhesions were handled, how much more rapidly the operation was done, also call to mind the fact that in the first instance both sides were diseased, while in this case only one side was involved, which, while not always the rule, may occur just as there may be a unilateral epididymitis in the male. You will then be able to form some idea as to the respective virulence of these two poisons. It is very rare that gonorrhœal infection leads to fatal peritonitis, though that it does occasionally do so there is in my mind no question. One other point to which I wish to call your attention, as bearing upon the infectiousness of these different organisms, is that with rupture of pus-tubes such as these, where every evidence points to their being gonorrhœal in character, no drainage is resorted to, we merely sponge out the cavity and close the incision just as if there had been

no rupture. The uterine packing in this case was removed on the third day, and a small strip of aseptic gauze reintroduced into the cavity. This was removed two days later, and aseptic vaginal douches given for a week to prevent any reinfection. This woman is now left with a practically healthy endometrium, with a uterus, as you would see by examination, normal in size, normal in position, and, more important still, with an appendage upon one side, possessing its sexual function and capable of bearing children and continuing a useful member of society.

Before leaving this case I would ask you to look at the temperature-chart, and you will see there has been some elevation of temperature towards the latter part of her convalescence. This was due to a stitch abscess, which did not show itself until after the suture was removed. Infection took place not at the time of introducing the suture, but probably in its removal, which will further impress upon you the great precaution that we must take in every detail of such an operation from its very beginning until the patient is up and about, until, in other words, she is perfectly well. While this stitch-abscess did not interfere with her convalescence, while it is not a dangerous thing, still it is annoying, and shows an imperfect technique in some part of our work.

CASE III.—This patient, aged thirty-four years, upon whom we will operate to-day, gives also a gonorrhœal history. She has never been pregnant, so she states. She denies ever having had an abortion, but her occupation (prostitution) very naturally makes us doubt almost any statement she might make in regard to this. However, be that as it may, she gives a history of gonorrhœa with the usual sequelæ following this disease when involvement of the tubes occurs.

Upon opening the abdomen we find many adhesions, the intestines being closely adherent to the uterus and to the abscess sacs as we suppose them to be upon each side. Carefully separating these adhesions we come down to what we find to be a large ovarian abscess upon one side, and a double pyosalpinx. Whether there is any connection between the ovary and tube on the one side it is impossible for us at present to say, nor can we do so until we have removed the sac, opened it, and demonstrated the communication. We will proceed with what appears to be the most difficult part of the operation first,—that is, removal of the larger and more adherent sac. Carefully separating the adhesions, we find that the ovary is involved in



**FIG. 3.—CASE III.—Pyosalpinx, ovarian abscess, gonorrheal in origin.**



**FIG. 4.—CASE IV.—Bilateral pyosalpinx, puerperal in origin.**



the mass; that it seems to be completely degenerated. Notwithstanding the great care I have exercised, notwithstanding the fact that these adhesions are not relatively very tough, there is nevertheless a rupture of the ovarian sac. The other side we remove intact, the disease being confined to the tube, which we see, upon opening, is filled with a thick, creamy pus. The cavity is now carefully sponged out, irrigated with salt solution, only, however, at the point where the pus has come in contact with the structures, the general cavity having been shut off by flat sponges which we inserted before beginning the enucleation. We are sure there has been no escape of pus into the general cavity, and by our thorough washing and sponging we feel reasonably sure that all infectious material has been removed from the cul-de-sac and the exposed peritoneal surface. Believing this to be as I have stated of gonorrhoeal origin, we will close the wound without drainage, and we trust that she will go through a good convalescence and make an easy recovery. Opening the first sac removed, we find there is no communication between the tube and ovary. It is probable that the fimbriated ends of the tube spread out over the ovary, becoming adherent to it, allowing infection of the ovarian structure, possibly through a ruptured Graafian follicle, with the organisms causing this trouble; they find here a prolific soil for their rapid multiplication and development, and as they do so, encroaching upon the normal ovarian structure, enlarging the ovary gradually at the same time, until the normal stroma is entirely destroyed by a suppurative and thinning out process, which has taken place as a result of the distention with pus.

CASE IV.—The next case is one of exceeding interest, as you will see when the history is detailed to you. The patient is a young girl, aged nineteen years, who, as a result of an unfortunate indiscretion, became pregnant. Desiring, of course, to conceal her pregnancy, not wishing to go to full term and be delivered of a child, she had performed upon her—by whom I do not know—an abortion. You may in your practice, gentlemen, often witness just such cases. It is not for us to moralize, nor to deliver to you a sermon on morality, as we know, of course, that none of you in your future practice would be guilty of destroying a foetus. You will have, I am sure, in your work some woman of good family, some poor unfortunate girl, who, being unable to control her passion, or after per-



suasion by some gay Lothario, has tasted the forbidden fruit, pregnancy results, and she comes to you for relief. Unfortunate as it may be for her, as much as you may pity or sympathize with her, I am sure you will know what to do, or rather, I should say, what not to do. There are, however, persons, not doctors, in all communities possessed of little knowledge of these things, and yet enough to know how to produce abortions. The woman, as a result of their ignorance and through their dirty methods, is in imminent risk of losing her life. They know not what the word aseptic means, they carry on their dirty sounds and on their filthy tents all sorts of pathogenic micro-organisms, which find entrance into the uterus, become implanted upon the placental or decidual surfaces, and give rise to such a condition as we have in this patient. She is one of those unfortunates who sought relief from the filthy abortionist. What is the result? A puerperal sepsis, first somewhat mild in character, then of a most virulent kind. The abdomen became distended, pulse rapid, tongue coated, pain all over the abdomen, more severe in the pelvis. There was at first not much discharge, and what little there was, was not offensive in character. The infection was evidently one of the muscularis of the uterus, also of the tubes themselves from the start. She was curetted, irrigated, washed out, antiseptics were applied locally, etc., by the first attending physician. Another gentleman saw her and opened the cul-de-sac through the vagina, washing it out thoroughly. I had examined her previous to this operation and concluded at that time that she had pus-tubes. The uterus was fixed and tender, the abdomen still somewhat painful on pressure, though swelling and acute pain had subsided. This was a month after the first symptoms of sepsis developed. Following the second operation or vaginal incision with packing and drainage there was some slight amelioration in the symptoms. The temperature fell for a day or two, though it again went up. She then came entirely under my care, and in the hope, though at the time I thought it only a faint one, of retaining her tubes, and of being able to avoid any further operative procedures, I persisted for three weeks with packing, uterine and postuterine drainage, and hot vaginal douches. Finding at the end of that time that, instead of being better, she was worse, and that the accumulation in the tubes was, if anything, even more than at first, I decided upon an abdominal section. She herself, moreover, insisted upon having some-

thing, even if radical in character, done; she had suffered excruciating pain; she had been bedridden during her entire illness, and to-day we will try to give her the relief which she has demanded and which her condition imperatively calls for.

We find that the pelvis is filled with adhesions, which, as you see, are extremely difficult to separate. We must first separate all of these intestinal adhesions before we can reach the tubes. We now come down to a retroflexed uterus; we find the tubes passing directly behind the uterus, so that we must also separate the adhesions between the uterus and the sigmoid flexure before the tubes can be enucleated. Having now gotten the adhesions completely separated we find some pus, but only a small amount, coming up from the extreme depth of the retrouterine cul-de-sac. This we sponge out, and continue with much difficulty the enucleation of the tubes, which, as you will see, are not very large and do not contain much pus. Having separated them we now ligate and take off the ovaries as well as the tubular appendages. Everything being cleaned and thoroughly sponged out, we find that the pus which came up after separating the uterus was from a sac formed, as I can clearly see, by the uterus and the bowel above and the vagina below, at the point of the vaginal opening, which was made, as I have told you, some time since. This so-called pyogenic surface we will touch with carbolic acid, packing it off thoroughly; we will place a drainage-tube and gauze down into the cul-de-sac, and by this means shut off the cul-de-sac and its infectious points from the general cavity.

The operation, as you have seen, has been exceedingly tedious, as, you will remember I have so often told you, these cases of puerperal infection are. What the outcome will be it is impossible for us to say. We know the virulence of this character of poison, and unless by our packing we prevent contamination of the general cavity, it is quite probable that she will die with septic peritonitis. Her pulse is good, she is in good condition, and I hope she will make an untoward recovery.

[NOTE.—This last patient has completely recovered from the operation, although she now presents severe nervous phenomena due to the artificially produced menopause. Her physical health, however, is perfect.—L. F.]

## STERILITY.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY HENRY CLARK COE, M.D.,

Clinical Professor of Gynæcology in the Bellevue Hospital Medical College; formerly Professor of Gynæcology in the New York Polyclinic, New York City, New York.

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GENTLEMEN,—Although this is such a hackneyed subject, I venture to call your attention to it again for a few minutes because it is one which possesses especial interest for the general practitioner. The amount of literature bearing on sterility is enormous, from the classical work of Sims, which gave the first impulse to the study of this subject, down to the latest French monographs, with their exhaustive and often absurd details. Speaking of the work of Sims, I may be allowed to express the opinion that its influence is felt now almost as strongly as at the time it first appeared. A glance at articles in current medical literature will convince the reader that gynæcologists are always on the lookout for a purely mechanical cause for this condition, and are seeking to devise an equally mechanical way of overcoming it.

It would be out of place for me to enter into a detailed statement of the number of possible causes of sterility, since these are given at length in every text-book. Many of the factors—impotence in the male, dyspareunia, uterine displacements, etc.—are so obvious that no one could overlook them. I wish to lay special stress upon the influence of antelexion and stenosis of the os internum in preventing conception, and to inquire how far we can hope to successfully overcome it.

I venture to say that when a healthy woman, who has been married for three or four years, applies to a specialist to learn why she has never become pregnant, and the examination reveals the presence of a moderate degree of antelexion with an external os a little smaller than normal (the pelvic organs being otherwise in a

healthy condition), nine men out of ten will at once jump at the conclusion that they have found the cause of the sterility. They will recommend divulsion, with or without the use of a stem, and will make the most confident promises as to the result of this operation. Now, I would caution you against such a hasty action; it is unscientific and unfair to the patient.

The subject is not so simple as it would appear at first sight. Who, I would ask, is in a position to affirm positively just what degree of narrowing of the cervical canal is necessary in order to present a positive obstacle to the entrance of the spermatozoa? Every gynaecologist of wide experience has seen women conceive with an os so small as barely to admit a probe; on the other hand, women whose pelvic organs are as normal as any represented in an anatomical atlas, who have no history of any local trouble, and who menstruate without pain, remain childless throughout the course of a long married life. In other words, the causes of sterility do not always lie on the surface. I do not refer to those peculiar cases in which both husband and wife are perfectly healthy, and perform their marital duties in a regular manner, and yet in whom conception fails to occur, although when either of these parties marries again, the union is fruitful. These involve unknown causes, or at least they lie outside of the sphere of medical science.

The first duty of the physician in investigating a case of sterility is to ascertain whether the wife is really at fault, yet too often this is the last information which is sought for. Many poor women are subjected to a long course of treatment, perhaps to repeated divulsion and curettage, incision of the cervix, and even abdominal section, when they are really not at fault. In the absence of any obvious cause for the sterility on the part of the female, the condition of the husband should be carefully inquired into. This is sometimes a difficult and delicate matter, especially in this country where people are more sensitive about these things than they are abroad. A German or a French physician would not think of beginning treatment in a case of sterility without first examining the husband, looking carefully into his past history, especially as to attacks of gonorrhœa, etc., and finally requesting a specimen of semen for examination. I do not know why we should not be equally sensible in America. If the condition is sufficiently serious to require medical and surgical treatment at all, the physician certainly has a right,

and the wife has a right, to know who is the offending party. It is my own custom to request an interview with the husband, to state the matter frankly to him, and to say that I would like a specimen of semen.

Twice during the past few days I have been placed in such a position. A lady came to my office who had been married for ten years, yet had never been pregnant. She was extremely anxious to have a child. Her own history was negative; she had always been perfectly well; she had no dysmenorrhœa, and, on examination, I found the usual condition,—slight antelexion and a certain amount of stenosis of the os externum,—although I was able to pass a small sound without much trouble. I explained the situation to her, and asked her if her husband was healthy. She replied in the affirmative, and said that the sexual act was perfectly normal. I then told her that I saw nothing in her condition to constitute a bar to conception, and asked if the husband would not be willing to submit a specimen of semen. He came to my office and we had a frank talk. He had had two or three attacks of gonorrhœa in early youth, but was positive that he had never had epididymitis, single or double. He said that he was perfectly competent to perform the sexual act. Following my directions, he brought me specimens of semen on two different occasions, which were quite fresh and had been preserved at the normal temperature. After the most careful examination I was unable to detect a single spermatozoön, and naturally gave an unfavorable prognosis. The patient was willing and anxious to have an operation, but naturally, under these circumstances, it was not to be thought of. In another nearly similar case active spermatozoa were found two hours after intercourse, so that I felt justified in recommending an operation.

Less manly was the conduct of another husband who brought his wife to me for examination because he had been married for two or three years and she was still childless. She was a perfectly healthy woman and had an entirely negative history. On examination I found her pelvic organs typically normal, and stated frankly that I saw no reason, so far as she was concerned, why she should not conceive. I afterwards ascertained that her husband, although a man of considerable intellectual power, and apparently healthy, was absolutely impotent. He had never been able to perform the sexual act satisfactorily, and, from his wife's description, had only

an imperfect ejaculation of a thin fluid, unlike semen. And yet this man would have cast the entire blame upon his wife, and doubtless would have allowed me to operate upon her had I seen fit. It is rather a sad commentary on our sex that we are so apt to forget the sins of our youth and to cast the blame on the innocent. Men find it convenient to overlook early indiscretions, and even when such important issues are involved cannot recall former attacks of epididymitis which have undoubtedly rendered them sterile. I need not remind you that azoöspemia in the male is a very different condition from absolute impotence. A man may be perfectly healthy, he may perform the sexual act in an entirely satisfactory manner, and yet his semen may be absolutely sterile. There is no way of ascertaining this except by a microscopical examination. It would seem as if this should constitute the first step in the treatment of every case, and yet how seldom do we think of it, and how often do we blame the wife! It seems to me that with a little tact this matter could be arranged without offending the *amour propre* of the husband or the delicacy of the wife. We can thus ascertain positively if there is a prospect of anything being gained by local treatment.

Another point to be borne in mind is that because a woman has been married a year or two, and has not conceived, she is not to be regarded as absolutely sterile, and at once subjected to operative treatment. It is well known that many women remain sterile three, five, or even eight years, and then bear two or three children, so that we must be extremely careful how we condemn our patients to a life of barrenness because of the presence of some fancied abnormality. I do not believe that in the case of a healthy woman who has been married only a year or two, whose husband is healthy, and who has no local abnormality, any treatment whatever should be instituted. Time should be allowed to elapse; the patient should be encouraged, and not led to believe that she is necessarily to be childless because she has not conceived during the first year.

When a patient comes to your office asking you why she cannot bear children, go over her history very carefully. I do not mean the ordinary history of obstructive dysmenorrhœa with which you are so familiar, and which we always associate with ante flexion and contracted os, but a close comparison of her condition before and after marriage. We have had a number of young married women here who told pretty much the same story. They were strong,

healthy servant-girls, without an ache or a pain; they menstruated normally and hardly knew when the flow began. They married, and from a month or two after the marriage they had seldom been free from backache, leucorrhœa, and painful menstruation. Sometimes there is a clear history of gonorrhœal infection, but more frequently we suspect, rather than actually ascertain, that their troubles are due to sexual excess during the early months, or to the prevention of conception. Apropos of this latter subject, I would advise you to read the classical chapter in Goodell's lectures, in which he points out in striking terms the evils that may result from this practice. Such women may escape the acute inflammatory troubles, and may present none of the gross lesions of sufferers from gonorrhœal infection, but the large, heavy, tender uterus and the constant profuse leucorrhœal discharge are sufficient explanation of the reason why these unfortunates when they wish to become pregnant find that they cannot. I emphasize the importance of inquiring pretty closely into the habits of patients, and of seeking to trace out the causes of ill health in many of these sterile women. I have already alluded to the fact that a marked degree of ante flexion and stenosis of the os externum do not constitute an absolute bar to conception, and that many of these patients may, under more favorable conditions,—improvement in their general health, change of scene, temporary separation from their husbands,—conceive and bear children in spite of the apparent mechanical obstruction.

Now, with regard to the examination. If it is entirely negative, tell the patient so frankly; do not seek to exaggerate a slight ailment. The uterus may be of normal size and in its normal position; there is no tenderness over the ovaries, no leucorrhœal discharge; and yet a careful review of the history will elicit the fact that immediately after coitus a peculiar spasmodic contraction of the vagina occurs, and all the semen is expelled. This has seemed to me the only explanation in several cases.

The general health has not a little to do with sterility. In a certain class of patients who, while still quite young, suddenly take on adipose, begin to menstruate at irregular intervals, or profusely, and at times have almost absolute amenorrhœa, the prognosis is not entirely bad, because under dietetic treatment, the reduction of fat, and proper exercise, menstruation may return, and they may subsequently conceive.

But there is another cause of sterility upon which too little stress has been laid. It is only since we have performed so many abdominal sections that we have learned that numerous slight adhesions may fix the ovaries and tubes in abnormal positions; may cause occlusion of the distal end of the tube, and yet give rise to no symptoms. The patient menstruates normally and is free from pain, yet when we expose these organs at the operating-table their condition is found to be such as to absolutely prevent the passage of ova into the uterus, or the progress of spermatozoa through the tubes. Now, these are conditions which cannot be ascertained either by the eye or by the skilled touch. Many of these patients frequently give no history whatever of any attack of peritonitis. I have frequently questioned such women closely, and they have denied ever having been confined to bed, even for a single week, yet there has evidently been at some time a localized peritonitis, presumably, though not necessarily, of gonorrhœal origin. This only emphasizes the fact which I have stated before,—i.e., that the causes of sterility are often hidden.

This leads me to say that the prognosis should always be guarded, since we do not know in many cases why an apparently healthy woman, with a healthy husband, does not conceive, and we are not in a position to promise a positive cure by any course of treatment or operation. You can hardly take up a medical journal without reading an enthusiastic article regarding some new method of treatment, or some new device for curing sterility. Numbers of intra-uterine stems have been invented, each of which has its ardent advocates. The general practitioner, on reading the long lists of successful cases, is apt to infer that the sole cause of sterility is obstruction of the cervical canal, and that the direct and simple cure consists in maintaining the patency of this canal. In many instances the patients have been married only a year or so, and the operator was not in a position to affirm that they would not have become pregnant without such mechanical aid. In others, the true cause has been, not the mere dilatation of the cervical canal, but the renewal of the endometrium after curettage. You know how common conception is immediately after an abortion, and how prone patients with incipient cancer of the uterus are to conceive. This is simply another illustration of the fact that when the uterus is more congested than normal, and its endometrium is in a state of hypertrophy,



or is undergoing regeneration, it seems to be more receptive to the impregnated ovum than at other times.

Accordingly, I would advise you to exercise the greatest discretion in the matter of prognosis. Provided that you have satisfied yourself that the fault does not lie with the husband, and you find a local condition in the female which you regard as a sufficient explanation for her failure to conceive, be careful how you promise an absolute cure from any method of treatment or operation. Not only may you cause a fresh disappointment, but you do not add to your own professional reputation by promising too much. In my experience the best plan is to be absolutely frank. Tell the patient exactly what you find,—your own doubts, if you have them. Explain to her that by performing a certain operation you place the uterus in the most favorable condition for conception; that you are unwilling to state positively that conception will occur, but that it does follow the operation in a certain proportion of cases. Having done so much you will feel, and she will feel, that everything possible has been accomplished to favor the wished-for result.

I need not describe the operation with which you are so familiar. You have seen it done many times here, and you know how it should be done, and with what precautions. Personally, I cannot say that I have met with brilliant results. In a few cases—less than half a dozen—I really feel that pregnancy was directly due to divulsion and curettage. In others, conception took place so long afterwards that I felt doubtful whether it was due to my treatment or to an improvement in the patient's general health.

I have referred only to the commoner factors in the causation of sterility. It is self-evident that a retroflexed uterus must be replaced, that a gonorrhœa must be cured, a neoplasm removed, or any other of the grosser lesions which prevent conception attended to. Having divulsed and curetted the uterus, and maintained the patency of the canal, either with gauze, with a stem, or by any other method that is preferred, can we go a step further? I have already alluded to the presence of slight, impalpable adhesions. When these are suspected, especially if there has been a previous history of gonorrhœa or pelvic peritonitis, and, above all, if the patient suffers with more or less persistent colicky pain showing attachment of the bowel, I would regard it as perfectly legitimate in selected cases, after divulsion and curettage, to open the abdomen, or to perform vaginal

section, to break up these adhesions, free the uterus and tubes,—not removing healthy organs,—catheterize the tubes, or even resect them if necessary, and thus place the entire genital tract in the most favorable condition for conception. In a woman whose only symptom is sterility, who has never had any history of trouble within the abdomen, who is entirely free from pain, dysmenorrhœa, or other pelvic trouble, this might be regarded as radical treatment; so that I would prefer to limit it to cases in which there is an indication of adhesions, if not of actual disease, of the adnexa.

Reference is often made, especially in French text-books (in which this subject, often treated with almost prurient details, seems to be a favorite one) to artificial impregnation. It is hardly necessary to say that, in spite of the experience of Sims, this *dernier ressort* will hardly obtain favor among us. To the physician, as well as to the patient, it is a most disagreeable procedure. I have never resorted to it but once, and then only at the urgent solicitation of a woman upon whom I had performed various operations without success. After several trials, I thought that I had been successful. The patient did miss two periods while absent from the city, but I was unable to determine positively whether she actually had an abortion or simply had suppressed menstruation. The operation is so distasteful—not to say disgusting—that it is doubtful if you will ever be called upon to resort to it.

I have sought in this fragmentary way to simply touch upon the practical points of a large subject, leaving you to fill in details at your leisure from the text-books. There are certain facts upon which I have placed special emphasis, and they are these: A patient often applies to you on account of sterility alone. In such a case, before beginning the treatment, note her history thoroughly; ask to see the husband, and, even at the risk of incurring his displeasure, explain to him that it is necessary to demonstrate positively whether his semen contains spermatozoa or not before proceeding further. If he is really anxious to have children, he will co-operate with you; if not, he will usually be unwilling to have you operate upon his wife, especially if you cannot promise a cure. To the popular medical mind, the cause—I might say the only cause of sterility—is anteflexion and stenosis; in other words, an obvious mechanical obstruction to impregnation. Specialists of equal skill and experience will differ as to the degree of obstruction sufficient

to cause sterility and as to the indications for operative interference. When there is a well-marked dysmenorrhœa, it is much easier to decide. Then we have two objects in view,—not only to overcome the sterility, but to relieve painful symptoms. In every case we should give a guarded prognosis. No special method of operating or special method of treatment offers any particular advantage over another. Over-enthusiasm is to be viewed with suspicion in every branch of medicine, especially in gynæcology. The surgeon who cures every case is, to say the least, exceptionally fortunate. A small proportion of patients will undoubtedly be cured by a purely mechanical operation, but in a large proportion all that we can promise is that we have met obvious indications, and have placed the uterus in a favorable condition for conception, but that we do not guarantee that this will occur.

I might mention an interesting case in my own experience which shows the possible dangers of conception after a long period of sterility. It is a well-known fact that such patients are most prone to ectopic gestation, probably because the tubes have been partially occluded by a former inflammation. Several years ago I assisted a friend in an operation upon a lady, sterile for nine years. She had an acute antelexion with stenosis, for which she had long been treated. We divulsed and curetted. Two months afterwards she became pregnant in the left tube. The pregnancy advanced to the fourth month, the tube ruptured, and she died. This is not the only case of this kind which I could mention; therefore, I would add the caution that it might not be the most desirable thing for a woman who has been sterile for a number of years to become pregnant, especially if she has passed her prime.

# Ophthalmology.

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## THE OPERATIVE TREATMENT OF ECTROPION OF THE EYELID.

CLINICAL LECTURE DELIVERED AT THE ROYAL LONDON OPHTHALMIC HOSPITAL.

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GENTLEMEN,—There are few affections more disfiguring than that produced by exposure of reddened congested conjunctiva in ectropion of the eyelids. Not only is the condition a very unsightly one, but from the constant overflow of tears through eversion of the puncta it often causes great discomfort, and from the unnatural exposure of the eyeball is frequently attended by considerable danger to that organ.

Fortunately, the affection can in the majority of cases be relieved by operation, but to obtain success care has to be taken to select a procedure suitable to the case, and attention has to be paid to details in the manner in which it is carried out. I do not in this lecture propose to enter into the lengthy process of describing all the operations which have been practised or suggested for ectropion of the eyelids, but to point out the general principles which should guide a surgeon in the choice of operation in any particular case, and to quote some illustrative cases in which I obtained permanently good results.

The degree of ectropion for which operative interference may be required varies considerably. It may be that only the edge of the lid is turned out and a narrow band of thickened congested conjunctiva is exposed; on the other hand, the margin of the lid may be so drawn down that the whole conjunctival cul-de-sac is everted. These more severe cases are the result of cicatricial contraction following destruction of a portion of the skin, while the slighter cases

may be due simply to chronic thickening of the margin of the lid and spastic contraction of a portion of the orbicularis muscle. In some of the worst cases of cicatricial ectropion the margin of the lid becomes actually lengthened from the traction which is exerted, so that to restore it accurately to its normal position a wedge-shaped piece of the lid adjoining the outer canthus has to be removed.

In the treatment of these cases of cicatricial ectropion, to make good the loss which has occurred some new skin has to be implanted. In some a flap can be twisted into position from a neighboring part to which a pedicle of attachment is retained. In other cases the amount or position of the cicatricial tissue is such as to preclude this, and the question then presents itself, Is it possible to transplant, so as to retain its vitality, a piece of skin completely separated from some other part of the body of sufficient size to fill the raw surface which has been left? That this can be done, producing a permanently good effect, I am able to prove to you by quoting the following case:

Henry W., aged eight, when a year old fell on a stove and burnt his right cheek and lower lid. He was admitted to the Moorfields Hospital on October 5, 1895. There was then much eversion of the right lower lid, the conjunctival surface of which was drawn away from the globe and somewhat thickened. Cicatricial tissue extended downward from the margin of the lid for about an inch and a half, below which point the skin was loose and freely movable. A V Y operation was performed without, however, producing any permanent improvement in the condition.

On January 11, 1896, the patient was re-admitted into the hospital, when there was said to be marked ectropion of the lower lid, which was somewhat thickened, and the everted conjunctiva was congested. Two days later, on January 13, a plastic operation was performed, a flap of skin measuring about two inches by one inch being transplanted from the forearm. On February 6, twenty-three days later, it was noted that the flap had quite healed, that it was in a good position, and that there was no ectropion.

He was seen again on July 25, 1897,—i.e., a year and a half after the operation. There had been no contraction of the flap since he left the hospital, and the lid remained in good position.

Whether the flap which is to be inserted in these cases is taken from some neighboring part and remains connected with a pedicle

or is completely separated, the first stage in the operation is the same. It consists in making an incision through the skin a short distance from the margin of the everted lid and parallel with it, the object of the incision being to free the edge of the lid and allow of its being drawn into contact with the opposite one. The edges of the lids should then be stitched together, the stitches being passed through the free margin of the tarsus on each side so as to obtain a secure hold. Horsehair is the best material to use for these stitches, as it can be kept in longer, having less tendency than silk to cut its way out. When the margin of the displaced lid has been freed and united to the one opposite, the size of the flap required to fill up the raw surface left can be estimated. As it is important to have the flap fitting as accurately as possible when it is simply laid on the wound, it is well to cut out a pattern of it in paper first. It is necessary to cut the flap larger than would appear to be required, because the skin shrinks a great deal directly it is detached from the surface. The flap should consist of skin only, it being dissected up cleanly so that no fat or areolar tissue is left adherent. Before the flap is applied to the raw surface on the lid, care should be taken to see that all hemorrhage from the latter has been arrested. The flap should be laid on the raw surface, but no sutures should be passed through it, which is a most important point in insuring its vitality. In several cases in which stitches have been inserted I have known large portions of the flap to slough, the sloughing always commencing around the point of insertion of the sutures. In the case above narrated the flap was retained in position merely by the firm application of the dressing, which was not removed until a week after the operation. In the next case I am going to describe to you the flaps were held in position by a method suggested by Argyll Robertson, in which stitches were passed across but not through them. A stitch is first inserted through a small fold of skin at one angle of the raw surface, when it is carried diagonally across the flap to the opposite angle and passed through a small fold of skin in the same way. Next it is carried across the flap vertically and tethered again, and finally it is passed diagonally across the flap to the angle vertically opposite the one at which it was first inserted, where, after being tethered through a fold of skin, it is finally tied.

David Z., a Moor from Tangiers, aged about forty-five, was admitted as a patient at the Moorfields Hospital on July 14, 1897.

Eleven years previously he had had an extensive burn of the right side of his face. There were large areas of cicatricial tissue on the right side of his forehead, nose, and brow. His right upper and lower lids were both much everted, the ocular and palpebral conjunctivæ were injected, and the right cornea was clear and unaffected. The day following admission, the patient having been placed under ether, incisions were made parallel to the margins of each lid, which after a little dissection were freed and united with sutures. Two oval pieces of skin, each about an inch and a half long and an inch wide in the broadest part, were then placed on the raw surfaces left in the upper and lower lids. These were secured in position by Argyll Robertson's "tethering" sutures. The dressing consisted of iodoform powder, green protective, and allembroth wool. On July 18, three days later, when the dressing was first removed, the lower flap was found to have completely united, but there was some slight sloughing at the outer end of the upper one. The patient unfortunately had to leave the hospital sooner than I wished, and as he has returned to Morocco, I have not seen him since. On the day of his discharge, July 29, fourteen days after the operation, the grafted skin looked healthy and the lids were in good position, but much thickened.

In these two cases the transplanted flaps of skin were taken from the inner surface of the patient's forearm. The skin there is smooth, free from large hairs, and easily accessible. I think, however, in any future case I shall take the skin from the inner side of the arm, a position in which the somewhat large cicatrix which is necessarily left would not be so likely to become exposed to view. In the first case I mentioned, what is called a *V Y* operation had been tried and proved unsuccessful. In cases of cicatricial ectropion, however, in which the amount of eversion is less extensive, it often produces very satisfactory results. The following are brief notes of two cases in which I performed this operation in the early part of 1896.

Alfred J. N., aged sixteen, as the result of a scald with boiling water when nine years old, had his right lower eyelid drawn down. On March 2, 1896, a *V Y* operation was performed on his right lower lid. Twelve days later the following note was made: "Flap has taken well; lid still slightly everted, but tissues have not quite flattened down." I wrote asking him to come and see me a short

time ago, and received a letter in reply saying he was unable to do so, as he had last August enlisted in the army, which is good evidence that the operation had proved successful.

William J., aged fourteen, came to the hospital with the inner half of his right lower lid everted and the exposed conjunctiva red and thickened. He stated that when he was three months old he had an operation performed on his right lower lid for a nævus, and that since then it had become drawn downward. On April 30, 1896, a *V Y* operation was performed on his right lower lid. On May 14 the following note was made: "Flap has healed by first intention except just at the apex, where it has sloughed. Skin-grafts placed here. Lid in better position; very slight ectropion." I saw the patient again on March 17, 1898, when there was very slight ectropion and no everted conjunctiva, but the punctum lacrymale was slightly drawn away from the globe. The patient did not complain of any discomfort, and there was practically no disfigurement remaining.

The *V Y* operation is only suited for cases of ectropion of the lower lid. In performing it a V-shaped incision is made below the everted lid, its apex being downward and in a line with the centre of the part most everted. The flap included in this incision, composed not only of skin but of the subjacent tissue also, is dissected up sufficiently to allow the margin of the lid to be raised readily into its normal position. The edges of the wound from which the V-shaped flap has been cut are then drawn together and united from below upward, as far as may be thought necessary, by sutures or hare-lip pins. The wound, which was originally the shape of a V, is thus converted into that of a Y. A few sutures are required to keep the flap in position, but it is better not to insert any near the apex, because if it is subjected to much traumatism it is liable to slough, as it did in the last case. If there is any difficulty in securing the apex of the flap in position, a "tethering" suture can be passed across it somewhat in the way already described. In one case I used it with a satisfactory result.

Cases of ectropion the result of spastic contraction of the orbicularis muscle, or of the slight amount of cicatrization which occurs in marginal blepharitis, require operations of a less extensive character than those just described. The insertion of what are known



as Snellen's sutures often produce excellent results in these cases, of which the following is an example:

Roland H., aged fifty-six, a mariner, came to the Moorfields Hospital on January 6, 1896. The margin of his right lower lid had been everted and inflamed for ten years, and that of his left for from five to six years. I applied the galvano-cautery to the inner surface of the left lower lid along its margin, hoping that the resulting cicatrization would tend to draw it inward. On January 16 I inserted two Snellen's sutures in the right lower lid. Three days later these sutures were removed, when the lid was in good position and its margin was no longer everted. At that time the left lid, in spite of the cauterization, was still everted, so three Snellen's sutures were inserted into it also. A small abscess formed in the track of one of these after it was removed and had to be punctured. When the patient was last seen, about a month after the operation, the eversion on both sides was quite cured.

To apply a Snellen's suture, a piece of silk is taken threaded at each end with a curved needle. The needles are inserted a short distance, four to five millimetres, from one another through the conjunctival surface of the everted lid at the highest point of the everted part. They penetrate the tarsus and are carried on beneath the skin to about the level of the margin of the orbit, where they are made to emerge through the skin and are tied over small pieces of drainage-tube. Two sutures are thus inserted at the junction of the inner and middle and outer and middle thirds of the lid. By traction on them the degree of inturning of the margin of the lid which is required can be regulated.

# **CATARACT OPERATIONS; MULES'S OPERATION ILLUSTRATED BY SKIAGRAPHS; CAPSULOTOMY; OPERATION FOR PTERYGIUM.**

**CLINICAL LECTURE DELIVERED AT THE MEDICO-CHIRURGICAL COLLEGE.**

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GENTLEMEN,—CASE I.—This young girl is seventeen years old and is suffering from congenital cataract. She was brought from an institution for the blind to this hospital with the hope that an operation could be performed upon one or both eyes and vision given her. A careful test has been made with a lighted candle in the dark chamber of the ophthalmoscopic room, and it was found that she has good projection of light in both eyes, for had she not had the visual power to locate the glare from the candle it would have been useless to have attempted the operation. I have frequently called your attention to this test, as it will be useful in examining patients and in giving a favorable or unfavorable prognosis before an operation. As this patient has good projection of light in both eyes we may assume that the retina is in a good condition, and that if no inflammatory reaction follows the needle operation a favorable result may be anticipated.

Another test which may be applied to this patient is to see whether the irides respond promptly to light. This is done as follows: I cover both eyes with my hands. This places both eyes in a darkened chamber. The pupils dilate. One hand is suddenly removed from the eye, and the iris, which was dilated, now suddenly contracts, caused by the flood of light falling on the retina, and by reflex action contracts the pupil if the retina is in a normal condition.

The cataracts in this patient are somewhat different in consistency to those usually found in the congenital variety. Upon close inspection both lenses are shrunken and chalky. But what is very peculiar is that the equatorial diameter is about normal, while the antero-posterior has greatly shrunken, giving the lumen the appearance of white chalky scales suspended in the pupils. In the many cases that I have examined I have never seen anything like this before. The glistening bits of cholesterol crystals shining among the marble-like deposits of the lens make it a most unique picture.

*Operation.—Discission.*—A puncture is made with a Bowman's stop-needle in the lower and outer quadrant of the cornea, midway between the centre of the pupil and the corneo-scleral margin. Two sweeps of the point of the needle are made in the vertical and two in the horizontal directions, to lacerate the capsule of the lens. We follow this by stirring the central anterior portion of the lens. This causes rapid breaking down of the cataract and allows its particles, which become separated, to pass into the anterior chamber and later to become absorbed.

One thing I must repeat, and that is, be careful not to do too much in the stirring up of the lens. Absorption takes place in from three to nine weeks. Sometimes a secondary operation must be performed. It is not necessary for me to caution you about the sterilization of all instruments, atropine solution, and bandages used at the time of the operation and subsequently.

We are indebted to the late Dr. J. C. Saunders, the founder of Moorfields Eye Hospital, London, for having suggested the operation. He also invented the delicate needle with which he so successfully operated. Sir William Bowman many years afterwards modified the shape of this needle by having a shoulder placed on it. Nearly all ophthalmic surgeons use it, and it is now known as Bowman's stop-needle. I pass it round for inspection.

When the posterior capsule remains intact and obscures central vision a secondary operation must be performed,—capsulotomy. I prefer making an incision in the cornea with a broad needle through the capsule in the vertical direction, then passing de Wecker scissors through the corneal opening, allowing the blades to separate, one blade to pass behind the capsule, the second to the front horizontally, and with one snip cut the capsule in two. This operation is most

successful. The after-dressing in this operation is the same as that followed in senile cataract.

The only complication which may arise is the too rapid swelling of the lens. This pressure on the ciliary bodies may cause cyclitis. When it does, it may be necessary to remove part of the soft lens by Teal's method,—that is, make an incision into the anterior chamber and suck out part of the lens substance with a specially devised instrument.

CASES II. and III.—These two patients are subjects of the same malady, senile cataract. Both patients, one sixty and the other sixty-five years of age, are practically blind.

This disease of the crystalline lens occurs in individuals usually past the meridian of life. It was known to the ancients and was removed by an operation, evidence of which is shown by old writings. Eye specialists flourished in the city of Alexandria when that city was the centre of civilization. It was also known to the Chinese, and is probably one of the oldest operations of which we have authentic data. The ancient operation was known as the couching or reclining method. A needle was passed into the eyeball posterior to the ciliary bodies. The point of the needle passed forward to the lens, which is then dislocated backward and downward into the vitreous. It was not very successful. Secondary inflammations frequently followed, and the eye was lost.

To-day an incision is made in the cornea, part of the iris removed or not, according to the will of the operator, and the cataract pressed out through the corneal wound. There are many different methods, all more or less similar, and all having the same object in view. To Daviel, who in 1745 made the first corneal incision, are we indebted for the operation upon which all subsequent methods are based. I shall follow in part a method suggested by Professor Snellen,—that is, to make a semiflap incision in the cornea, inclining the cutting edge of the knife backward as I approach the apex of the cornea and make a large incision in the conjunctiva, which gives me a conjunctival flap. An iridectomy is also performed. In rupturing the capsule of the lens I use Jaeger's cystotome, it being less dangerous than the ordinary straight cystotome commonly used.

The delivery of the cataract is done by slight pressure upon the lower half of the cornea, tilting the lens forward, accentuating the

pressure with the Daviel spoon as the lens makes its appearance in the wound. Great care must be taken to see that all cortical matter is cleaned out of the anterior chamber and that the iris does not become incarcerated in the lips of the wound. The conjunctival flap is then stroked into its place and the eye bandaged. The after-dressing in cataract operations is a very important factor in its successful termination. Every ophthalmic surgeon has his own peculiar way of applying a dressing to an eyeball. The method adopted in this school is as follows: Immediately after the wound is closed a few drops of atropine solution are dropped into the eye, followed by mild irrigation of solution of hydrargyrum bichloride 1 to 2000. The eyelids are closed, and over this is placed two or three drachms of sterilized vaseline, and all closed with eye-pads held in place by adhesive plaster. I have used such a dressing for the past fifteen years most successfully. This dressing is replaced in twenty-four hours. The large number of cases dismissed from the hospital in nine or ten days proves that the method is a good one.

*CASE IV.—Staphyloma of the Cornea, a Sequela of Gonorrhœal Ophthalmia.*—This child; twelve years of age, through a very unfortunate infection, became the subject of gonorrhœal ophthalmia, in which the right eye was lost, although the most careful treatment had been instituted. As I told you in a former lecture when speaking on this subject, when you are dealing with an infection of this kind you have to do with a poison almost as deadly as nitric acid. It will destroy a cornea in three days. This case comes to you as a warning to notify all patients who have this terrible disease that they should be as careful as if they were handling the most deadly poison. The whole of the corneal tissue has been destroyed, and this large projection is called a staphyloma. It is a mass of cicatricial tissue, and possibly its bulging forward is the effect of intra-ocular tension, as the eyeball is very hard to the touch. In point of fact, we have as a complication glaucoma, and the abnormally high tension causing the great pain which is present day and night. As the eyeball is very much disfigured and painful, its removal has been advised. Instead of removing the eyeball I shall perform a Mules's operation. Dr. Mules has given the following table, which I will read to you, showing the advantage of this operation over that of enucleation:

ENUCLEATION	<i>versus</i>	MULES'S OPERATION.
1. Complete removal of globe and its contents.		1. Retention of the framework of the eye.
2. No stump, therefore sunken eye.		2. A firm, round globe forming perfect support for artificial eye.
3. Disturbance of all muscular relations and arrest of movement.		3. Perfect harmony of muscular movement retained.
4. A fixed staring eye attracting attention.		4. Fitted with selected eye defies detection.
5. Patient shuns society.		5. No qualms as to personal appearance.
6. Arrested development of orbit in case of children.		6. No interference with growth of orbit.

You have frequently seen me perform this operation, and while every operation is not successful, I must repeat what Mr. Carter, of London, says about it: "I am performing it with increasing pleasure on every available occasion."

In those cases which have not been successful I am sure something must have been amiss with my technique or the sterilization. I count those cases failures in which the stitches gave way and the glass ball came out. Even when this takes place, the clean sclerotic coat which closes up makes a better support for the artificial eye than a complete enucleation. Some ophthalmic surgeons are still unfavorably disposed towards this operation, but I predict that the day will come when it will be universally adopted. Mr. T. Herbert Bickerton, of Liverpool, always speaks and writes about it most eulogistically, and Mr. Brudenell Carter, of London, is quoted as saying, "I may say that I have totally abandoned enucleation, except for glioma in children for cases of injury in which sympathetic ophthalmia has actually commenced, or for cases of injury or of painful complete glaucoma in aged and feeble people, in whom appearance is comparatively unimportant. Except in these conditions, I regard enucleation as a slovenly and unjustifiable mutilation which no surgeon has a right to inflict upon a patient."

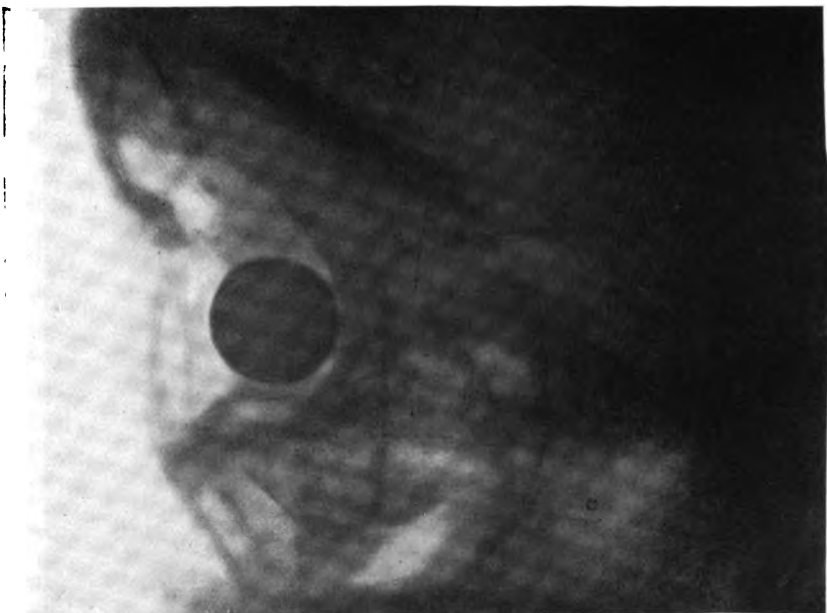
I will repeat the method of the operation. The eyelids are separated with the ophthalmostat. The conjunctiva is dissected off from its corneoscleral attachment back to about the equator of the eyeball, the muscles also being separated; then the cornea is excised. This is best done with a large Beer knife, as if performing a flap operation for cataract. The lower half of the cornea is removed with curved scissors, and the contents of the globe are taken out with a small scoop devised for the purpose.

Great care is necessary to remove the ciliary bodies and choroid and the head of the optic nerve, leaving a clean, white sclera. Mr. Carter has devised a rubber bulb which is inserted into the scleral cavity and inflated with air to produce pressure on the central artery to prevent hemorrhage. As this application has not been a success with me, I pack the scleral cavity with sterilized gauze. After waiting a few minutes this is removed, and the contents of the scleral cavity are again thoroughly irrigated with antiseptic fluid and again packed. A sterilized glass globe, which is best suited to the case, is then inserted with a specially devised instrument. The sclera is split vertically so that the edges may be drawn together and held by stitches of No. 4 black silk, using large needles, completely hiding the glass ball. The orbit is again thoroughly irrigated with the hot solution and the socket packed with sterilized gauze. The bandages are not disturbed for three days. During this time the patient may complain of considerable pain. It is better to control the pain with an opiate than to disturb the bandages. This constant pressure keeps down the conjunctival swelling which I formerly experienced, and was a factor, I believe, in causing the silk threads to cut their way out and ultimately to allow the glass or silver ball to escape.

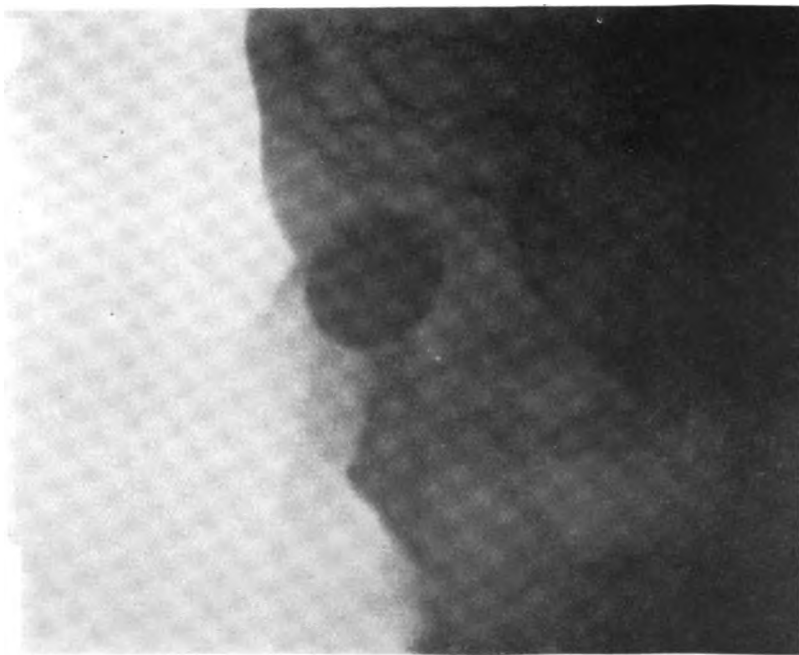
As a rule, the conjunctival sutures are not removed under six or ten days. It is important that both eyes are kept bandaged for at least six days. By allowing the liberty of one too much rotation of the eye is permitted and, as a consequence, the antagonistic muscles of the operated eye pull apart, and there is great pressure brought against the sutures, which are liable to be torn out, a probable cause in several of my cases.

From the large number of operations now under observation, and no unfavorable ones reported, it may be considered a very safe one, and if we have in evisceration a method equally as safe as in enucleation, why not give the patient the advantage of the new method?

To confirm my experience I quote still further Mr. Carter, who says, "I have not seen a single instance in which there has been any evil consequences, or in which I have had occasion to wish that I had done something else. The fact that some injuries of the eye are followed by sympathetic ophthalmia in its fellow, and that this may entail total loss of sight, should never be absent from the



**FIG. 1.—Skiagraph by direct rays, showing circular shadow of silver ball in Mules's operation.**



**FIG. 2.—Skiagraph by oblique rays, showing elliptical shadow of silver ball in Mules's operation.**







FIG. 3.—Sklagraph by direct rays, showing circular shadow of a glass ball in Mules's operation.



memory; but, at the same time, it affords no excuse for the sacrifice of eyes in circumstances in which the danger does not exist or in which it can be obviated or overcome. To indiscriminately remove injured or inflamed blind eyes can hardly be called either science or surgery, for it is clearly the duty of the surgeon to be as conservative as circumstances will permit, and to preserve as much of any organ or endowment as may be possible."

The skiagraphs which I show you were taken by Dr. Furbush, and they show you the location of the glass ball and silver balls in the orbit. No. 1 and No. 2 are silver balls; No. 3 is a glass ball. The operation in the case of No. 1 was performed October 12, 1897; the other two at a later date. The photographs show the artificial eyes in place. No one suspects that these patients are wearing artificial eyes, and further, that they never have a sensation of discomfort from them. Several of my old patients come to see me and they make the same report.

CASE V.—*Capsulotomy; Secondary Cataract.*—This patient was operated upon for senile cataract two years ago and was able to follow her ordinary household duties until quite recently, when she noticed that her vision was growing dim. Upon examination I found a gradual thickening of the posterior capsule which obstructed her vision. She now seeks surgical aid. I shall perform a capsulotomy. Many surgeons tear the capsule with a Bowman's stop-needle, but I prefer a different method. I take a broad needle, make a vertical incision on the outer side of the cornea, and pass the point of the blade through the capsule close to the pupillary margin of the iris, which has been dilated with atropine. I then insert a de Wecker's scissors and pass the one blade posterior to the capsule, and the second in front across the anterior chamber directly in the centre of the pupillary area, and then make the cut. The capsule separates, and usually leaves a very large clear pupil.

The usual cataract dressing is carried out, and at the end of a week the patient is sent home.

In all cases where an incision has been made in the cornea and capsule, the patient must be warned not to use his eye too much for close work until all evidence of irritation has disappeared. The vision will be corrected with the usual cataract glasses.

CASE VI.—*Pterygium.*—This man has had the growth which you see over his eye for some time, but it has only been for the last

few years that it has been of any annoyance. There is a constant irritation, burning and itching, as the patient expresses it. I shall remove it. There are several methods for its removal, but here the transplantation method will be followed, and when properly performed the growth does not return. This is done as follows: An incision is made in the conjunctiva above and below the growth, along its borders from the cornea to the caruncle. The conjunctiva slightly dissected above, but very extensively below the growth, making a pocket which extends to the insertion of the inferior rectus muscle. The pterygium is then separated from the eyeball with scissors, leaving, however, the corneal attachment intact, and then raised, and a needle (two needles are threaded on one strand of silk) is passed through the corneal end of the pterygium from the upper side downward; the second needle is passed in like manner, but brought out upward, leaving enough tissue between the two threads that will not cut through; with the strabismus hook the pterygium is separated from its corneal attachment (Prince), and is then turned downward into the cul-de-sac, the needles, one at a time, passed through the conjunctiva near to the insertion of the inferior rectus muscle, bringing the head of the growth almost in contact with the muscle, being held in place by the silk thread which is tied. This brings the raw surfaces of the pterygium against the raw surfaces of the eyeball. The edges of the conjunctiva are brought together over the pterygium by three strands, and while there is some puckering of tissue in the caruncular space, it usually disappears, making a successful operation.

The after-dressing will be the same as that followed in the ordinary conjunctival or strabismus operations. The stitches are removed after four or five days.



**FIG. 4.—Right eye, Mules's operation.**



**FIG. 5.—Left eye, Mules's operation.**



# **A SALUTARY OCULAR REFLEX; ABSORPTION OF CATARACTOUS DEPOSIT IN LEFT EYE FROM IRIDO-CHOROIDITIS IN THE RIGHT.**

CLINICAL LECTURE DELIVERED TO THE GRADUATING CLASS.

**BY WILLIAM CLARENCE BOTELER, M.D.,**  
of Frederick, Maryland,

Late Professor of Ophthalmology, Otology, and Laryngology in the Medical Department of Kansas City University, Kansas City, Kansas.

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GENTLEMEN,—Since the development of the science of ophthalmology in the early part of this century, the most perplexing question that has confronted its investigators has been the nature and path of transmission of pathological processes from one eye to another. At the present time casualties of this nature are classified under the head of sympathetic ophthalmia. In the earliest textbooks on ophthalmology they were ascribed to “metastasis;” later, reflex processes were thought sufficient to account for them; and at the present time, as stated above, the term sympathetic ophthalmia comprehends the entire class.

The designation, sympathetic ophthalmia, had its origin probably in the supposed instrumentality of the sympathetic nerves and ganglia; but from the work of modern pathologists being confined to tracing the connection through, first, the optic nerve, then the anterior ciliary nerves, and at no time through the sympathetic, we feel justified in suggesting that if the term “sympathetic ophthalmia” is to have a fixed place in ophthalmic nomenclature, and the agency of the sympathetic nerves is to be acknowledged thereby, we must concede the possibility of the occurrence of other reflexes than irido-cyclitis and those pathological in their nature, or possibly the elaboration of salutary conditions.

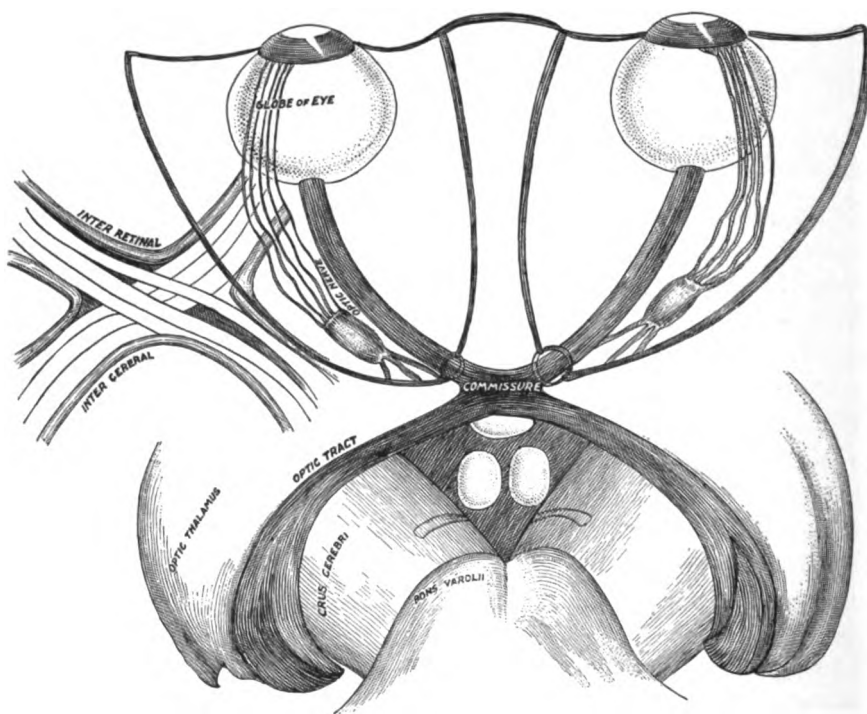
The sympathetic is essentially a circuit for nutrition, and primarily transmits trophic influences unless its centres are injured or its anastomosing and intercommunicating fibres are obstructed or



destroyed. To demonstrate the practicability of an unusual regenerative phenomenon among ophthalmological reflexes, I beg the liberty of referring to several charts executed and especially designed to make this question plain.

Chart No. 1 (executed for me by one of your number) is a view of the base of the brain, showing the eyeballs, optic nerves, the optic commissures, the geniculate bodies, and, in the more minute dissection at the side, the arrangement of the decussating fibres that

CHART No. 1.



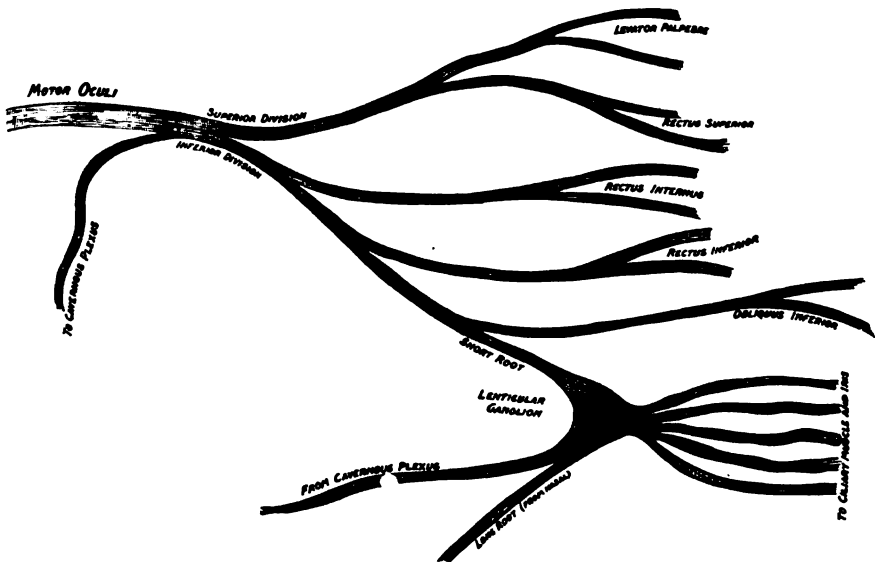
are concerned in crossed impressions. The heavy line impressed upon the chart marks a sectional view of the outline of the bony orbits, showing the aperture of entrance of the optic nerve; the dark quadrangle and filaments at the side are the lenticular ganglion and the fibres of the short ciliary nerves.

In Chart No. 2, we have the subdivision of the third or motor oculi nerves and the short ciliary nerves that extend to and supply the iris, ciliary muscle, ciliary bodies, and all the parts of the uveal

tract. It is plain to see that there is a tract of direct communication through the inter-retinal fibres of the optic nerve between the two eyeballs; there is also in each of the ocular orbits a lenticular ganglion and ciliary nerves; so you might hastily conclude that there should be little difficulty in explaining what we stated above was a "perplexing question."

Ophthalmologists first fell upon the idea that reflex or sympathetic ophthalmias were transmitted "through these inter-retinal fibres of the optic nerve;" but on this theory optic neuritis must exist, and so many cases were adduced to show that the optic nerve

CHART No. 2.



was not at all involved, and that the optic nerve had no direct terminal connection anatomically with the ciliary region from whence sympathetic ophthalmia emanated, that as a tract of communication it was for years entirely abandoned.

As the ciliary region or uveal tract, where traumatism originates this grave disorder, is supplied entirely with the ciliary nerves, this course was next selected; but as the ciliary nerves of the two orbits have no direct connection with one another, the investigator was again perplexed by a missing link. With the sentiment of investigators divided between the one and the other tract, ophthalmologists stand to-day involved in vague uncertainty as to which is right, so

many varied phases of the pathological common reflexes occurring that now seem to favor one and now the other course. It is my impression that neither course as described above is adequate to explain the transmission of all the reflexes from one eye to another, but that some are transmitted by one tract, some by another, and some require the combined use of the optic and ciliary distributions, and some the optic, ciliary, sympathetic, and trigeminus.

There is sometimes found a small fibre of the ciliary nerves that penetrates the optic with the arteria centralis retina, establishing a circuit of transmission not usually understood. It is this communicating circuit that I want to bring to your notice, as it is the only unbroken circuit of transmission that anatomists have as yet presented that will fully explain the usual phenomena of sympathetic ophthalmia.

Prof. J. B. Wolfe, ophthalmic surgeon, Glasgow, in his excellent work on ophthalmology, 1882, Lecture VIII., dwells extensively on the subject of sympathetic ophthalmia, and possibly outstrips all his distinguished competitors in the accuracy of his conclusions as to the medium of transmission of both degenerate and reparative reflexes. He says: "The sympathetic action seems to be set up through the agency of the fifth nerve in conjunction with the sympathetic. The fifth nerve is a nerve of sensation and nutrition, and the irritation of it is quite sufficient to account for the reflex phenomenon." He speaks of how the fifth nerve and the sympathetic interlace, and regards them both as participating in sympathetic ophthalmia. He then cites the phenomenon in traumatic tetanus where the irritation of a sensory nerve, even of the cerebro-spinal system, has the property of transmitting pathological conditions from the periphery to the brain, where it is further elaborated and reflected. His argument is the best to-day before the profession; but it is weak in that he gives no reason why only morbid and not salutary processes may be transmitted, nor why it should be reflected into the other eye and not to some other part of the nervous system.

Schmidt Rimpler in his text-book of 1889 speaks only of "the transmission of morbid processes." He does not suggest even the remote possibility of the transmission of reflexes of a conservative nature; he does not attempt to elucidate the mysterious tract nor the philosophy of the process. He seems to ascribe all that there is in sympathetic ophthalmia to the transmission of cyclitis, and does

poor justice to the usual profundity of German pathologists by his purely recitative treatment of the subject.

Soelberg Wells in his valuable treatise on the eye, third American edition, 1880, like Rimpler above, recites only the possible transmission of retrograde processes, going into detail concerning the various diseases and injuries to the ciliary region of one eye that may be transmitted to the other. He says, "There is no manner of doubt that this is done through the medium of the ciliary nerves," but his argument falls short in that sympathetic ophthalmia or reflex ophthalmias occur many years after the eyeball and the ciliary nerves at the source of irritation have been enucleated or destroyed; and he admits no place in his argument for the reflex transmission of salutary conditions.

Ernest Fuchs, of Vienna, in his late practical work, a recognized text-book, discusses the question as follows: "In what way the transference of inflammation from one eye to the other takes place has not been definitely settled. The ciliary nerves of the two sides are not connected with each other directly, as are the optic nerves, hence the transmission of inflammation in such cases cannot be considered a direct one. On the contrary, we have to assume that the ciliary nerves from an inflamed eye cause an irritation of a nerve centre which is transmitted like a 'reflex' to the ciliary nerves of the other side and to their termination in the other eye." But he does not state whether the centre referred to is that of the fifth nerve, the optic nerve, the sympathetic, or what centre, and makes no mention of the twig of the ciliary connecting it with the optic.

To be understood by you all, I am fully aware that medical writers in presenting text-books cannot go further into pathology than to elucidate the varied manifestations that occur to them. I am fully aware that no human mind can compass the many wonderful phenomena that may present in any country, and it is not my inclination to recite the above distinguished authorities in a critical manner. I have laid before you what they say and what they have left unsaid for the mere purpose of setting forth the peculiar case that I will now report, in contrast with the observations and experiences of these great seers who have gone before. *Contrast* gives identity to any subject.

In the case we have to show is a phenomenon declared to be im-

possible by an oculist of note in Kansas City. In it we have an illustration of the transmission of ocular reflexes not necessarily morbid, and even the possibility of transmission from a morbid process in one eye of conservative reparative processes to the other. Paradoxical as these assertions seem, the patient is an intelligent gentleman and still lives to corroborate me. I anticipate that the particulars which here follow will open wider the field of experiment in ocular reflexes, and I am free to say I believe it will help to bring investigators to a uniformity of opinion as to the tract.

This gentleman, Mr. Joseph B., of St. Joseph, Missouri, whose case is before you, applied to me first November 18, 1886, for the removal of senile cataract. He was about sixty-four years old, then in fair bodily health, had been a merchant and bookkeeper, and is a gentleman of unusual intelligence. He possessed the peculiar condition, monoblepsis, being absolutely at his advanced age myopic in his left eye and presbyopic in his right; whenever he undertook to read or write, one eye had to be shut. Thus, in approximating subjects closer to get a better image on the retinae, he had so strained the accommodation of his left eye as to have cut off osmotic nutrition to the lens and produced its death, or cataract. The lens seemed thoroughly opaque, and the operation was done, with the assistance of Dr. J. H. Stringfellow, at St. Joseph, Missouri, without any untoward incident. During the night of the third day before the corneal section had thoroughly united my patient fancied in a dream that he was enjoying a siesta in the mellifluous air of an orange-grove in Florida; that a mosquito the size of a terrapin crawled up on his eye, as the vulture descended on the liver of Prometheus, to eat it out. "I will catch you, old fellow," said the dreamer,—“knock you senseless with one blow.” The annihilating blow was delivered, but only to remind my patient that he had thumped himself in his tender unhealed eye. The gentleman's convalescence was retarded, but his vision, with a two and one-half cataract glass, was sufficient for reading during the daytime.

In the course of a few weeks the appearance of "secondary capsular cataract" began to develop. On examination I found remains of the posterior segment of the capsule obstructing. It was repeatedly needled; but vision was later so interfered with as to make it impossible for the patient to read at night, and with difficulty during the day, with that eye. Fearing the possibility

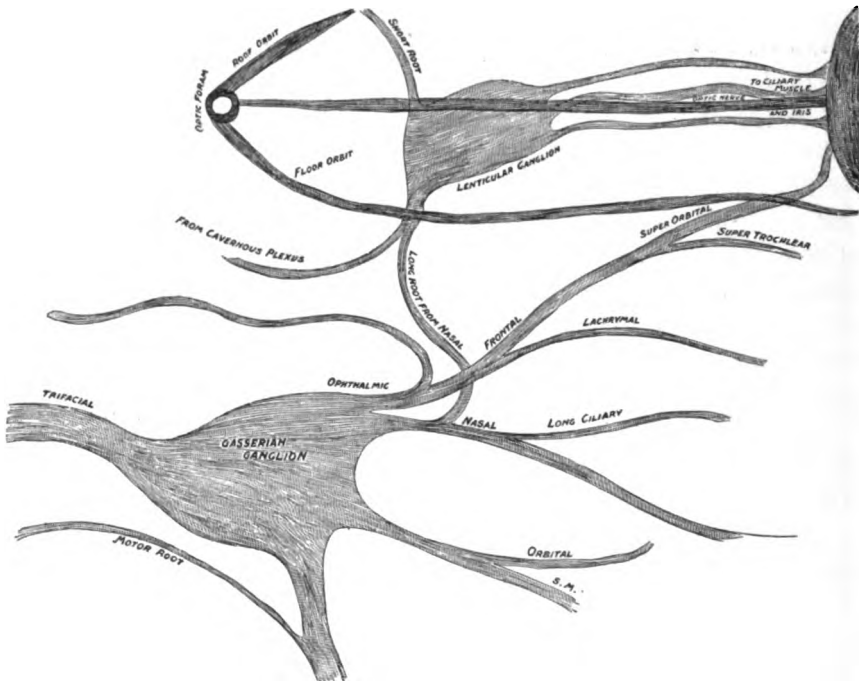
of setting up destructive inflammation in one or both eyes, I discontinued the needling, not entirely satisfied with the case. At the time of the cataract extraction above from the *left* eye, incipient cataract had commenced in the right eye. It slowly progressed towards total blindness until, from some cause not known, in the last week of October, 1896, an acute attack of iritis came on. The right or "far sighted" (presbyopic) eye was then entirely blind. He applied to me here for relief. *He states that he noticed before the treatment of the right eye and the subsequent removal of its cataract, coincident with the appearance of the irido-cyclitis, which I found it was, that the secondary cataractous film existing for eight years in the left eye had commenced to disappear.* The iritis was the result of an intumescent, pultaceous, over-ripe cataract pressing upon the ciliary region, obstructing the outflow through the canal of Schlemm in the right eye. After treatment and the subsidence of the ciliary inflammation, which had existed then nearly four weeks, it was found that the eight-year secondary cataract had been absorbed from the other eye and vision was perfectly normal. After complete freedom from ciliary irritation, the large over-ripe cataract, pressing on the ciliary region much as a foreign body, was removed, and he had normal vision with glasses in both eyes.

We have in this case the remarkable phenomenon of cyclitis in one eye from practically a foreign body (the dead and swollen lens) producing what in the other eye? Not destructive cyclitis or "sympathetic ophthalmia" in the other eye, but a regenerative or benign process,—i.e., the removal of a secondary cataractous deposit.

The transmission must have been through the ciliary nerves from the very nature of things, as they supplied the iris that was inflamed. The anastomosing branches of the trigeminus and sympathetic must have intervened, for the presence of a sensory afferent and efferent nerve is essential where trophic influence is to be exerted and hyperæmia established to effect nutrition, and from the close, uniform, unerring absorption of the secondary deposit, until it was all removed from the other eye, it could scarcely have occurred through any other circuit. I therefore feel that the pathological phenomena and physiological links in this case justify the conclusion that trophic influences can be transmitted, as well as morbid ones, from one eye to another, and that the chain of association will be found to be from the inflamed ciliary region of the

injured eye through the ciliary nerves to the lenticular ganglion, thence through the long root of the nasal and the ophthalmic branch of the fifth, the Gasserian ganglion, the fifth itself, to the floor of the fourth ventricle, to the centre, reinforced by the shorter circuit from the lenticular ganglion to the cavernous plexus of the sympathetic, and back to the branches of the short ciliary nerves. This seems to be a comparatively new channel of transmission only, in

CHART No. 8.



fact, hinted at by previous investigators; but I believe the manifestations in this case justify the conclusion that this very unusual reflex followed this course. As I have stated before to this class, the mere possibility of this occurrence was disputed by an oculist, hence I have searched all available records, literature, and citations for some corroborative instances. I have been unable to find anything in any modern text-books, but in a number of an early work on ophthalmology, published in America in 1846, nine years before Williams, the first oculist, began to practise in Cincinnati, nine years before the great discovery of the ophthalmoscope by Helmholtz, at

page 217 of the second edition of Dr. S. Littell's first work on ophthalmology he says: "A curious and instructive case recently occurred at Wills Hospital, Philadelphia, Pa. A patient was admitted with rheumatic iritis of the right eye who twenty years before had lost the sight of the left from a wound. The ciliary nerves had been injured by the accident; the pupil became oval and deviated towards the circumference of the iris. Previous to his admission he was barely able to distinguish light from darkness, but during the inflammation of the other eye (the last) the vision of the former gradually improved and was entirely restored *without any obvious cause*. When discharged from the house he could see equally well with both eyes."

There is a wonderful coincidence between the particulars of these two cases, although they were fifty years apart, and the only two that I know of on record. In both cases from trauma the left eye was disabled and certainly secondary deposit formed; in both cases the restorative emanated from an iritis in the other eye; in both cases absorption took place in the left eye from an inflammation in the right, reversing the order in "sympathetic" ophthalmia; and in both cases both eyes recovered.

To investigators this *newer Salutory Reflex* and possible tract is now promulgated, and I hope will receive the careful study of ophthalmologists and yourselves, as it seems to me a subject of considerable importance.



# Laryngology.

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## SUPPURATIVE INFLAMMATION OF THE FRONTAL SINUSES: ITS ETIOLOGY AND TREATMENT.

CLINICAL LECTURE DELIVERED AT THE MANCHESTER EAR HOSPITAL.

BY W. MILLIGAN, M.D.,

Hon. Surgeon to the Manchester Ear Hospital; Lecturer upon Diseases of the Ear in the Owens College, Manchester, England.

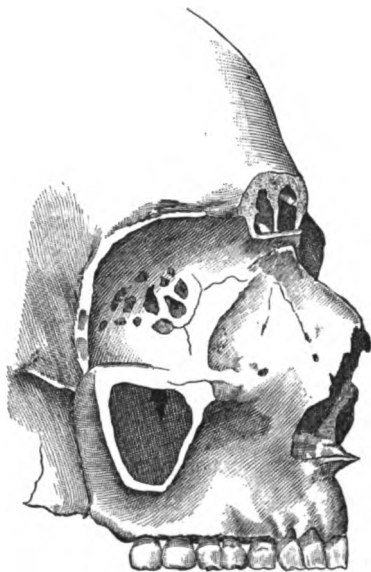
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GENTLEMEN,—During the past few years very considerable attention has been paid to the subject of suppurative disease of the nasal accessory sinuses. These sinuses, as you are aware, are the frontal, the ethmoidal, the sphenoidal, and the maxillary. Each has its own independent opening into the corresponding nasal chamber. The openings of the frontal, the anterior ethmoidal, and the maxillary sinuses lie in fairly close relationship to one another in the anterior portion of the middle meatus, whilst those of the posterior ethmoidal and the sphenoidal sinuses are to be found in the posterior part of the superior meatus. In cases of suppurative disease, pus coming from any of the first three named sinuses will be seen by anterior rhinoscopy in the middle meatus of the nose; whereas, pus coming from the last two mentioned sinuses will be found upon posterior rhinoscopy presenting in the superior meatal region and trickling downward and backward into the postnasal space. In actual practice, the difficulty which we have to face is the differential diagnosis between affections of the various sinuses,—a problem rendered specially difficult by the fact that there are frequently no very leading symptoms present which would serve to separate an affection of any one sinus from an affection of another. This difficulty of differential diagnosis is also accentuated, owing to the fact of the openings of the various sinuses being in such close proximity

to one another that it is at times very puzzling to say from which sinus pus is actually flowing.

To-day I cannot attempt more than a survey of the main symptoms and signs which lead to the diagnosis of a frontal sinusitis and of the various methods of treatment which are in vogue for combating this disease. The diagnosis and treatment of suppurative affections of other accessory sinuses must be left for consideration upon another occasion. The frontal sinuses (Fig. 1) are two in number, placed one upon either side of the middle line of the head immediately under the glabella. They may be symmetrical in size

FIG. 1.



Section of skull showing position of frontal and maxillary sinuses.

and shape, although great variations are known to occur without any external indication of this being so. In fact, it may be said that the external appearances of the part are no clue to the size or shape of the underlying sinuses. They are separated from one another by a median septum usually bony but at times fibrous, usually complete but at times perforated,—anatomical perforations being, however, very rare. Each sinus communicates with its corresponding nasal chamber by means of a somewhat tortuous passage, named the infundibulum, opening into the middle meatal fossa just in front of the bulla ethmoidalis, and in very close relationship with the

opening of the maxillary antrum and the anterior ethmoidal cells. In fact, the openings of the frontal and maxillary sinuses may be, and at times are, continuous with one another by means of a gutter-shaped fold of mucous membrane, a clinical fact of the greatest importance, as you will see presently. Occasionally the frontal sinuses will be found to communicate with the anterior ethmoidal cells of the same side, and so with their respective infundibula. In all cases the sinuses communicate directly or indirectly with the corresponding nasal chamber. The mucous membrane lining the sinuses is in direct continuity with that lining the nasal passage, but it is thinner, more closely adherent to the underlying bony parietes, and is paler in color. Bearing these few but important anatomical facts in mind, let us pass on to a consideration of suppurative disease of this sinus,—its etiology and its treatment. For practical purposes we may divide suppurative affections of this cavity into two main forms, acute and chronic, or, as it is more frequently called, latent empyema of the sinus.

Cases of acute suppurative frontal sinusitis are by no means frequent, and fortunately so, for the affection may be of such virulence as to threaten, if not to destroy, the patient's life. Most usually it is secondary to a previous catarrhal inflammation, to attacks of la grippe, to traumatism, to the entrance of foreign bodies, snuff, larvæ, etc., or to improper intranasal manipulations, obstructive nasal lesions, etc.

Its presence is ushered in by severe pain, at first local, but rapidly spreading over the head, swelling and œdema of the superimposed soft tissues, chemosis of the eyelids, high temperature, rapid pulse, etc. Should obstruction to the outflow of pus take place, distention of the sinus results, with bulging of its walls. Owing to the inferior or orbital wall being the thinnest, the effects of distention are first noticed here, while at the same time the eyeball is displaced downward and outward. If, however, distention should take place posteriorly, symptoms referable to increased intracranial tension will be noted, owing to the effects of pressure upon the anterior portions of the frontal lobes. Should this distention continue, perforation of the bony parietes may ensue, followed by most serious consequences. If perforation takes place towards the orbit, septic orbital cellulitis may occur, followed by loss of vision. If, again, perforation takes place posteriorly, septic pia-arachnitis or

extradural abscess may result. With regard to the treatment of this affection, no time should be lost in trying to allay the inflammation and in attempting to secure a return of free frontonasal drainage. Locally, three to six leeches should be applied over the sinus, or, where leeches cannot be obtained, an artificial leech may be employed. Ice-bags laid over the sinus afford a certain degree of relief. Intranasally, a spray of a four- or five-per-cent. solution of cocaine may be used, or a one-half-per-cent. watery solution of ichthyol, with the idea of relieving the accompanying oedema of the nasal mucosa, and so of promoting frontonasal drainage. Internally, a copious saline purge should be administered, while aconite, belladonna, or phenacetin may be given to allay headache, etc. Should no abatement in the intensity of the symptoms occur within forty-eight hours, I believe myself that the right thing to do is to open and drain the sinus. This may be accomplished either by means of a supra-orbital or a median incision, as I shall presently describe. When once the sinus has been opened and drained, resolution of the inflammatory process, followed by healing, rapidly takes place.

The more usual form of frontal sinusitis met with in practice is, however, the chronic form of suppurative disease, or, as it is more commonly called, latent empyema of the sinus. Latent empyemata, although by no means frequently met with, are, however, probably more common than statistics generally show; and I think that there can be no doubt that since la grippe has made its appearance a greater number of cases have been noted. No doubt in many cases latent empyema is a sequel to what had formerly been an acute or a semiacute attack of catarrhal sinusitis which had passed off, so far as all active symptoms were concerned, but which had left the mucosa in a vulnerable condition,—a condition, in fact, where pathogenic organisms found a suitable soil and multiplied with their usual energy. It may also follow obstructive nasal lesions, affections of other nasal sinuses, injuries, etc. The symptoms of latent empyema are not well pronounced, and frequently there is great difficulty in deciding whether the sinus is affected or not; in fact, in some cases, a positive diagnosis can only be arrived at after the performance of an exploratory operation; and, let me say, that I believe a straightforward external exploratory operation is not only quite justifiable, but is a much more rational and sensible proceeding than are many

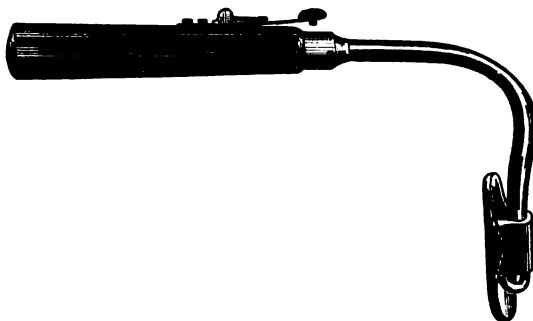
of the devices at present in vogue for intranasal exploration of the sinus.

Pain, which is such a prominent and diagnostic symptom in acute cases, is frequently entirely absent in chronic cases, or can only be elicited with difficulty. If pressure be made upon the inferior wall of the sinus (the thinnest wall) immediately above the inner canthus of the eye, a sensation of marked tenderness is at times complained of. A sensation of fulness and weight over the glabella is also a symptom of some importance. Darrack's symptom, or the sensation of pitching forward,—a sensation relieved by blowing the nose,—is described by a few patients.

The most important symptom or sign is, however, the presence of a purulent nasal discharge flowing from the anterior portion of the middle meatal fossa, to be seen upon anterior rhinoscopy oozing from the region of the infundibular opening. This discharge is usually increased when the patient is in the erect posture. The discharge is thick and creamy in appearance, and is at times very fetid. Again, anterior rhinoscopy will frequently reveal the presence of a polypoid condition of the anterior end of the middle turbinated body, and also at times an œdematous appearance of the septal mucosa in this neighborhood, both appearances probably being caused by the irritating qualities of the discharge. The practical difficulty, even when these symptoms or signs are present, is to localize the affection to the frontal sinus. My experience is and has been that, as a rule, the frontal sinus is rarely affected *per se*, but that other accessory sinuses—the ethmoidal labyrinth and the maxillary antrum more especially—are likewise simultaneously affected, and that, in fact, all three cavities may be suppurating at one and the same time. To exclude the participation or otherwise of the maxillary antrum in the suppurative process is by no means difficult; but it is exceedingly difficult, if not impossible, in many cases to decide whether the anterior ethmoidal cells are implicated or not. In order to exclude the participation of the maxillary sinus, it is only necessary, after having carefully washed out the nasal passage, to examine the cavity by means of Voltolini's (Fig. 2) or Escat's (Fig. 3) lamp. The examination is made in a perfectly dark room. Voltolini's lamp is introduced into the patient's mouth, and the mouth is then kept quite closed. When the current is turned on, the rays from the lamp pass through the trans-

lucent facial bones; but should the antrum contain pus the light will not pass, and hence one side of the face will be brilliantly lit

FIG. 2.



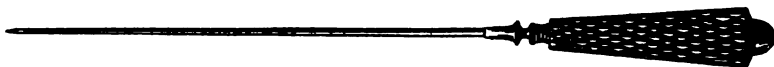
up, while the other, the diseased side, is opaque and dark. Escat's lamp is applied directly to the retromaxillary fossa, and shows very

FIG. 3.



distinctly any opacity from the presence of pus, etc. Should the cavity be found to permit of the passage of rays of light from the lamp,—in fact, to be translucent,—we may conclude that antral supuration is not present. Or, again, we may puncture the cavity by means of a Lichtwitz's trochar (Fig. 4) and wash it out. If pus

FIG. 4.



appears, we have positive evidence of disease; whereas, if no pus appears, we have equally reliable evidence that no disease is present. Unfortunately, we possess no such ready and reliable methods of ascertaining the presence or absence of pus in the ethmoidal labyrinth; but my experience has taught me that it is safer, when operating upon the frontal sinus, to clear away at the same time a considerable number of the anterior ethmoidal cells. This, I think, is not only advisable, owing to the fact that the two sets of cells

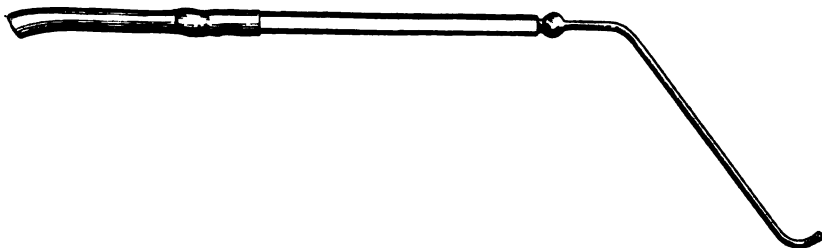
are so frequently found jointly diseased, but also because by so doing more room is gained for free frontonasal drainage, a point of the very greatest importance. The operative treatment of latent empyema has of late years undergone considerable modification, and in many respects considerable improvement.

Let us in the first place consider the various intranasal procedures which have been devised,—some of them of value, others of very questionable utility.

First of all let me direct your attention to the method of syringing out the sinus *per vias naturales* by means of a specially constructed canula.

The canula (Fig. 5) is passed upward into the middle meatus,

FIG. 5.



and in the first place a search is made with its point for the infundibular orifice. When this is found, it is gently pushed onward, and, by reason of its peculiar shape, it can undoubtedly, in some cases, be thrust into the sinus. Before actually passing a stream of fluid through the canula, I should advise you, in the first place, to blow in some air by means of a small rubber bag. Should this have the effect of driving any purulent material out of the sinus, there can then be no objection to washing out the cavity; but should the air stream not succeed in doing this I would caution you against using the fluid stream. In cases where the nasal mucosa around the infundibular orifice is œdematous or even polypoid, it is well to pin it down by cauterizing, either by means of an electric needle or by means of chromic acid, trichloracetic acid, etc.

It has been suggested that in cases of latent empyema benefit may be derived from amputation of the anterior end of the middle turbinated body,—anterior turbinectomy; the idea being that drainage is thereby facilitated. This may be so in some cases, but in

others I am convinced that it is of very little use; the obstruction to the outflow of pus being frequently situated at a much higher level than that of the middle turbinated body, being in reality situated at the opening of the infundibulum within the sinus.

Schäffer's method of pushing a curette forward and upward, in the hope that it may open the sinus, is a method I need only mention to condemn as being dangerous in the extreme.

In the great majority of cases where operative interference is required, you will, I think, find that some form of external operation is demanded. The more usual indications for operating may be summarized as follows: (1) retention of pus within the sinus; (2) the persistence of a purulent discharge from the sinus, with its accompanying symptoms and discomforts; (3) the presence of symptoms of cerebral irritation or of cerebral compression; (4) the presence of severe neuralgic pains, with impaired general health.

There are several methods of operating in vogue at present, but I propose to mention only one method, that usually now known as the Ogston-Luc operation.

A median incision is made, commencing at the root of the nose and carried vertically upward in the middle line from one and a half to two inches. The soft tissues are divided down to the bone, and are then powerfully retracted so as to expose an area of bare bone of about the size of a florin. Hemorrhage, which is usually very slight, is at once stopped by means of pressure forceps, etc. An opening may now be made into the sinus by means of a trephine or a gouge and mallet. If the trephine be used, the pin is placed in the middle line of the forehead, and a disc of bone (about the size of a sixpenny piece) is now carefully removed. The removal of this disc of bone will expose a portion of the mucous membrane lining both sinuses, an advantage in cases of bilateral empyema, but I think a disadvantage in cases of unilateral empyema. In unilateral cases I prefer to use a small gouge and carefully (and under good illumination) chip away layer after layer of bone until the mucosa is reached. In order to ascertain if pus be present, a fine probe should be thrust into the sinus and moved round and round. If pus be present, it at once begins to ooze upward by the sides of the probe, and in addition an escape of fetid gas usually takes place. The mucous membrane should now be freely divided and a careful examination of the interior of the sinus made by means



of good reflected light. At times it will be found thickened and granular, at times in a state of polypoid degeneration, and in still other cases pedunculated polypi will be found. All this diseased mucosa should be carefully and thoroughly scraped away, special attention being paid to clearing the infundibular orifice. A considerable portion of the floor of the sinus should be removed by means of a small gouge or sharp spoon, so as to make a large opening into the corresponding nasal passage. I am also in the habit of removing a considerable number of the fronto-ethmoidal and anterior ethmoidal cells, which, as has already been remarked, participate so frequently in the suppurative process. After all hemorrhage has been checked, the interior of the sinus should be swabbed with a solution of chloride of zinc (twenty grains to one ounce).

A drainage-tube with a specially constructed flange (Fig. 6) is

FIG. 6.



now slipped into the sinus and drawn down into the nasal passage until the flange rests upon the floor of the sinus, and is then fixed by means of a thread to the corresponding ala nasi. The external wound is now closed, care being taken to first of all accurately stitch the periosteum together, and then the superimposed soft tissues. A small pad of iodoform gauze is now put over the wound and an ordinary dressing applied.

Primary union takes place rapidly, and if the stitches be removed at the end of thirty-six hours the resulting scar becomes in a short time hardly perceptible. Subsequent irrigation is carried out through the tube in the nose. Many solutions are used for this purpose, such as carbolic lotion, boracic lotion, solution of iodoform in ether, etc., and in many cases it is necessary from time to time to alter the drug used. As the discharge becomes less and less copious, so should the irrigation become less and less frequent; but on no account should the tube be removed until the cavity has remained perfectly dry for at least ten days. Objections have been made to the median incision on the score that a considerable falling in of the tissues takes place and a corresponding amount of de-

formity follows, but if care be taken to accurately adjust the periosteum over the opening into the sinus, subsequent deformity can, as a rule, be obviated. The median incision has, I think, this great advantage, that when the sinus has been opened a much better view of its interior can be commanded than can be done when a supra-orbital incision has been made, and the further advantage, also, that the ethmoidal region is distinctly more accessible.

I beg, finally, to impress upon you the great necessity of (1) clearing away the whole of the diseased mucosa lining the sinus, (2) the necessity of securing and of maintaining free frontonasal drainage, and (3) the importance of patient attention to the numerous details in the after-treatment of these cases.

# Dermatology.

## BALDNESS: ITS VARIETIES, CAUSES, AND TREATMENT.

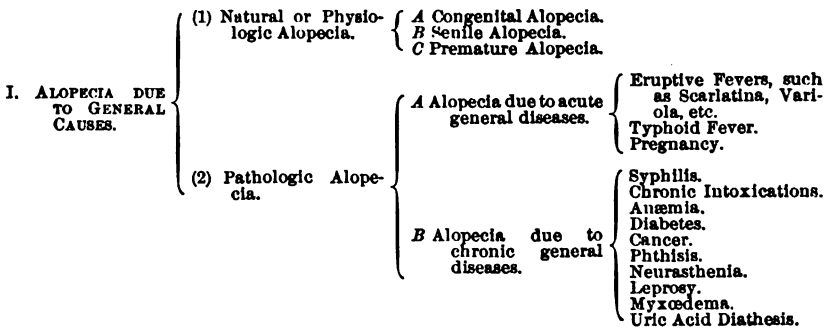
CLINICAL LECTURE DELIVERED AT THE PHILADELPHIA POLYCLINIC.

BY JAY F. SCHAMBERG, A.B., M.D.,

Associate in Diseases of the Skin, Philadelphia Polyclinic.

GENTLEMEN,—I have to-day the rarely afforded opportunity of presenting to you a number of patients exhibiting the various forms of alopecia. Physicians are constantly solicited by patients suffering from loss of hair to restore to them those ornamental appendages with which nature once endowed them. That baldness detracts from physical beauty all will concede, although the disfigurement is not nearly as great in males as in females. This subject, however, is not of importance merely on account of its bearing upon cosmetology. Hair-loss occurs as a symptom in many general diseases, in not a few of which it possesses certain distinctive characteristics which are of great diagnostic value.

The varieties and causes of alopecia are numerous. Brocq, in his "Traitement des Maladies de la Peau," has given a schematic classification, which I present to you in a revised and modified form:



II. ALOPECIA DUE TO LOCAL CAUSES.	{	(1) Diseases in which Alopecia occurs only accidentally.	{	Seborrhœa. Erysipelas. Eczema Seborrhœicum. Psoriasis. Syphiloderma. Folliculitis. Lupus Erythematosus.
		(2) Diseases in which Alopecia is a prominent symptom.	{	Alopecia Areata. Ringworm. Favus.

## ALOPECIA PREMATURA.

Alopecia prematura, or premature alopecia, is the form of baldness most frequently brought to the attention of the physician. This may commence at the age of eighteen or twenty, although it is more frequently observed about ten years later. It is characterized by a general thinning of the hair, which is most marked in certain regions. In perhaps the majority of cases the vertex is the part first and most extensively affected, producing a tolerably circumscribed baldness resembling the priestly tonsure. The frontal region suffers next in frequency. In women the temporal regions first show the thinning of the hair. Each combing and brushing is accompanied by a hair-loss calculated to excite alarm in the mind of the patient. The hair which falls is replaced by a finer crop, and this in turn by a still finer growth, until, after the lapse of some months or years, there remains nothing but a soft, downy, whitish fur upon the scalp.

Premature baldness has been attributed to a host of causes. Compression of the scalp by tightly fitting hats, prolonged intellectual labor, venereal excesses, bad hygiene of the scalp, the too frequent wetting of the hair, etc., have all been ascribed as fruitful causes. The most potent etiologic factors, however, are *heredity* and *seborrhœa capitis*.

Hereditary influence is so well recognized as a cause of baldness that even laymen read their futures in the shining cranial horoscopes of their fathers. Whilst premature baldness is hereditary in certain families, its transmission is not at all constant, many members escaping. Again, the heredity is often atavistic, skipping a generation.

Seborrhœa capitis, or dandruff, is responsible for a very large number of cases of premature baldness. The hair-follicle and the sebaceous gland are in such close anatomical relation that it is not surprising that disease of the latter should unfavorably influence

hair growth. In this condition the scalp is pale, and covered with small grayish or yellowish, dry or greasy scales. The hair becomes dry and brittle and falls out.

The treatment of premature baldness is largely prophylactic. Those who promise patients with partial or total "bald pates" a restoration of their hair are either ignorant of the course of these cases or are guilty of wilful deception. It is no more possible to regenerate hair from atrophied hair-follicles than it is to bring forth blossoms upon dead plants.

Prophylaxis should begin in infancy with the proper cleansing of the babe's scalp. The vernix caseosa should not be permitted to desiccate upon the scalp, but should be removed with soap and warm water after previous softening with some unguentous substance.

Women are much less subject to baldness than men. They, however, bestow more care upon their hair, do not wet it so frequently, and wear light and loosely fitting hats that permit of free ventilation of the scalp. The healthy scalp should not be washed more than once in two weeks; ordinary soap and water will suffice for this purpose.

Dandruff, or seborrhœa capitis, should never be allowed to go untreated. In this condition, treatment is directed towards the proper cleansing of the scalp and the stimulation of the sebaceous glands to healthy action. The tincture of green soap makes an admirable shampoo for the scalp, removing in a thorough manner the epithelial and sebaceous *débris* there present. The list of hair-tonics is legion. The following may be recommended as extremely useful:

R. Resorcini, ℥ii;  
Acidi salicylici, gr. xxx;  
Ol. ricini, f℥i;  
Spts. vini rect., f℥vi;  
Ol. bergamottæ, f℥i.

This is to be thoroughly rubbed into the scalp every night or every other night. The massage of the scalp produced by the friction is of decided therapeutic value.

In hair-loss resulting from other causes, preparations of a more stimulating character are often desired. For this purpose the following formula will be found most serviceable:

R Hydrarg. bichlor., gr. xii;  
 Beta-naphthol, gr. xl;  
 Ol. ricini, fʒi;  
 Spts. vini rect.  
 Spts. myrciæ, aa fʒiii.—M.

Sig.—To be rubbed in every other night.

Internal treatment is at times necessary to build up the general health, and so secondarily influence the nutrition of the hair. Arsenic, iron, strychnine, and cod-liver oil may be employed in appropriate cases. Nutritious diet, physical exercise, and fresh air will often accomplish more than the administration of drugs.

The prognosis of premature alopecia is always extremely guarded. Where an hereditary tendency to baldness exists, it is unfavorable. The most that can be accomplished in most cases is a lessening or cessation of the hair-fall for a longer or shorter period. In rare instances a certain amount of regrowth may be brought about.

As an illustration of the type of baldness just described, I present to you this young man of twenty-six years. You will note the very conspicuous thinning of the hair over the vertex and frontal regions. The patient says that he has been losing his hair for the past three or four years, but that it has become so much worse during the past few months that he has become alarmed. His father, the patient states, was quite bald at the age of thirty-five. The hereditary factor, you observe, is quite patent in this case. If you will examine the scalp, you will find that it is pale and covered with profuse grayish-yellow scales. Two causes are therefore operating here in the production of the alopecia,—heredity and dandruff. The first cause we cannot affect by treatment. The seborrhœa, or dandruff, however, is quite amenable to judicious applications. We will use here the resorcin lotion, the formula of which I have just given you. It is quite possible that this patient may return in a few days and tell us that the wash is doing him no good; that it is actually causing the hair to fall. Vigorous digital friction with any fluid will of course bring out a number of hairs; but hairs which fall upon such provocation are largely devitalized, and would fall later of their own accord. It is well in private practice, however, to inform patients in advance that the friction attending the application of the hair-wash will, particularly at first, cause the loss of a certain amount of hair.

I believe the prognosis in this case to be unfavorable. By removing the dandruff and stimulating the sebaceous glands and the capillary circulation of the scalp, we will in all probability be able to limit and delay the loss of hair. It would be unwise to promise any more than this.

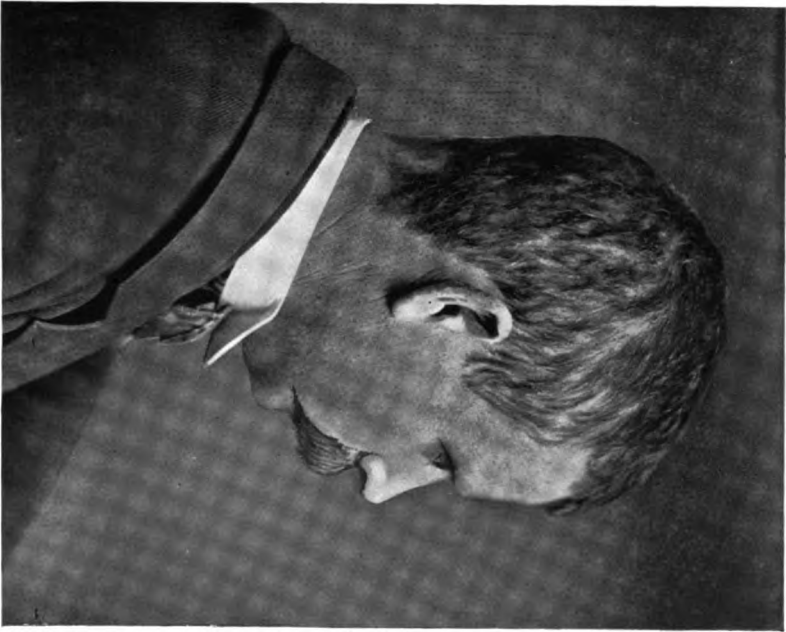
#### ALOPECIA SYPHILITICA.

The next patient is a robust mechanic of thirty years, who presents himself at the clinic for the first time. He complains that his hair is falling so extensively that the attention of his friends and acquaintances is being attracted. He noticed this hair-fall one month ago for the first time. The hair comes out in abundance upon the brush and comb, and upon the slightest traction. You see at once, gentlemen, a marked difference between the appearances of this and the first case shown you. Here the baldness is most conspicuous upon the lateral aspects of the head. Furthermore, it presents a peculiar streaky character, which contrasts strongly with the generalized thinning of the hair in the other patient. According to the history just elicited, this man had a chancre three months ago, which was followed six weeks later by an erythematous eruption upon the trunk and extremities. Inspection of the throat discovers the presence of superficial ulceration of the tonsils and mucous patches upon the palatal arches. We have here, then, a case of *sypilitic alopecia*.

The alopecia that accompanies secondary syphilis possesses such distinctive characteristics that it is often of great diagnostic value. Its conspicuousness renders it a most obnoxious symptom to the patient, who is most anxious to get rid of it on account of its compromising character. It usually makes its appearance from the third to the fifth month, at the decline of the early eruptive symptoms. The loss of hair may be so slight as to be scarcely noticeable, or, on the other hand, almost the entire hair of the scalp may be lost. In some cases the eyebrows, eyelashes, beard, mustache, and pubic and axillary hairs are affected.

Syphilitic baldness occurs in two forms,—one as a general thinning or extensive shedding of the hair, and the other, loss of hair in more or less circumscribed patches. Both of these varieties occur with about equal frequency.

The patchy form presents a curiously moth-eaten or mangy ap-



**FIG. 1.—SYPHILITIC ALOPECIA.**



**FIG. 2.—ALOPECIA AREATA IN MOTHER AND CHILD.**





pearance. The patches are irregular in outline, the hairs dry and lustreless, and the scalp scaly and unhealthy looking. Not infrequently the baldness occurs in irregular streaks, as is well illustrated in the accompanying photograph. (Fig. 1.)

The characteristic features of syphilitic alopecia are the extensiveness, rapidity, and irregularity of the hair-fall and the concomitant involvement of other hairy regions.

In rare cases the loss of hair is first, and even solely, observed in the eyebrows, in moth-eaten patches. This localization is considered by Fournier as highly characteristic of syphilis. In my experience, the loss of eyebrows and eyelashes has occurred with considerably more frequency in women than in men. Not uncommonly, this condition is the first to direct the attention of the patient to the fact that there is a deviation from health. In women, not only the initial sore but also the roseola may entirely escape detection. I will briefly narrate to you the history of an interesting case illustrating the diagnostic value of this form of alopecia.

K. M., a young woman of nineteen years, recently came under my care on account of loss of hair of the scalp, eyebrows, and eyelashes. The patient stated that she had had two years prior an attack of typhoid, which had caused a considerable thinning of the hair. Full restoration occurred in the course of four or five months. The present hair-fall dated back two months, and was rapidly progressing. Inspection of the scalp showed the hair to be greatly thinned, dry, and lustreless, the frontal and temporal regions being particularly affected. The eyebrows were largely gone, as were also the eyelashes. The loss of hair in these regions so greatly altered the appearance of the patient that many of her acquaintances failed to recognize her. Upon interrogation, I was informed that the hair of the pubic region had also been partly lost. The patient was sure that she had never had a sore upon the genitals or elsewhere, and that she had never noticed an eruption. She admitted, however, exposure to venereal infection. Examination of the body surface, throat, and vagina was negative. The inguinal glands were enlarged to the size of beans, and the post-cervical glands to the size of peas. The case presented a very pretty diagnostic problem. Was the hair-fall the result of faulty nerve innervation occurring two years after the attack of typhoid fever, or was it due to syphilis? In view of the remoteness of the attack of typhoid fever, and the simi-

larity of the alopecia to that seen in syphilis, the latter diagnosis was made. Although the patient has been under mercurial treatment now but a short time, there has been already marked improvement. The hair-fall has ceased and new hairs are beginning to grow in.

The prognosis of syphilitic alopecia is in general good; only exceptionally does it lead to permanent baldness. Restoration takes place rapidly under appropriate treatment.

I have thus far spoken of secondary syphilitic alopecia. It is only proper that I should call your attention to another form of alopecia occurring in this disease. This is the variety that follows ulcerating lesions of the hairy regions. It takes place later in syphilis at a time when tubercular and gummatous infiltrations occur. This baldness differs entirely from the early syphilitic alopecia. It is due to destructive ulceration of the hair-follicles, and is therefore permanent and irremediable.

#### ALOPECIA AREATA.

Permit me now to direct your attention to another form of alopecia quite distinct and apart from those previously considered. This form occurs in circumscribed patches, and is therefore designated *alopecia areata*. I have several patients here who represent different types of this affection. I will first show you the very rare phenomenon of a coincidence of this disease in mother and child. These patients have been under observation for some time, and their history is of sufficient interest to warrant its narration.

This child was brought to the clinic about ten months ago, at which time it was just three and a half years old. It then presented the appearances which you see well illustrated in this photograph (Fig. 2). The child was absolutely bald. Not only was there entire absence of hair upon the scalp, but the eyebrows, eyelashes, and lanugo hair of the body were completely wanting. The mother stated that the child had been born with a normal head of hair. The first manifestation of disease occurred at about the age of one and a half years, shortly after the hair had been cut for the first time. The mother noticed at this period several half-dollar-sized patches of baldness which persisted for about six months. Spontaneous restoration occurred, but was of short duration. In the course of two months the alopecia again developed, the patches spreading with such rapidity that in seventy days the child was totally bald.



**FIG. 3.—The same child four months later.**



**FIG. 4.—The same child ten months after inauguration of treatment.**



The patient was in this condition when he first came under observation.

I now show you a photograph (Fig. 3) representing the appearance of the case four months after the inauguration of treatment. You observe the calvarium is quite covered with hair which is an inch or an inch and a half in length. Here and there upon the parietal and frontal regions are small tufts of fine downy hair which indicate beginning regrowth in these areas. The eyebrows and eyelashes also show evidences of pilary activity. If you will now compare the photographs just shown you with the patient before you, you will be able to note the improvement that has taken place within ten months. The hair of the scalp has been almost completely restored. The entire scalp, with the exception of two small areas upon the temporal and occipital regions, is covered with a perfectly normal hair growth. The temporal and occipital patches are decreasing in size, and will no doubt soon disappear.

A particularly interesting feature of this case is that the mother is affected with the same disease. She has throughout her scalp a number of roundish patches, varying in size from a silver half-dollar to a dollar which are totally devoid of hair. It is necessary to separate the otherwise luxuriant hair in order to disclose the presence of these patches. The mother informs me that these developed at the age of fourteen. Being now thirty-four years old, you observe she has had this condition twenty years. She has neglected the treatment of her own scalp in order to be able to devote more time to the treatment of her child.

The child was placed on small doses of Fowler's solution from time to time, and used locally the following ointment:

R Beta-naphthol, grs. x1;  
Petrolati,  
Lanolini, aa ʒss.

This was rubbed into the scalp twice a day.

In view of the differing opinions held concerning the nature of alopecia areata, the occurrence of this disease in mother and child would seem to be of evidential value. Many of you are no doubt aware that the cause of alopecia areata has long been a mooted question. The French school emphatically declares this affection to be of parasitic origin. Indeed, so unanimously is this conceded

in France, that children affected with alopecia areata are detained from the public schools and, like ringworm cases, sent to a special school where such cases alone are received.

On the other hand, many distinguished authorities maintain that the disease is of nerve origin; in other words, a trophoneurosis. In confirmation of this view may be cited the experiments of Joseph, of Berlin, who succeeded in producing alopecia areata in cats by section of several cervical nerves.

The probabilities are that there are two varieties of cases, the one parasitic and the other trophoneurotic. Under no other assumption could the conflicting data advanced by both schools be explained. At first thought, it might appear that the case just shown you might throw some light on the subject. Upon reflection, however, you will see that, whilst the one school would cite it as an instance of contagion, the other would look upon it as evidence of the hereditary transmission of a neurotic tendency. Personally, I regard this case as a trophoneurosis. The rapid shedding of the hair of the scalp, eyebrows, eyelashes, and entire body suggests with much greater probability a dystrophy than a dermic contagion.

Alopecia areata, as the name implies, is a baldness affecting circumscribed areas. Only uncommonly does it become universal. I show you now a patient exhibiting a more common type of the disease than the preceding case. This man (Fig. 5), sixty years of age, presents, as you see, two circular dollar-sized patches of baldness on the top of the head. Over these areas the skin is entirely devoid of hair. The skin is pale, smooth, and soft, and slightly depressed beneath the surrounding healthy scalp. If you look closely, you will see that the follicular orifices upon these patches are less prominent than normal. The patient states that he has had this condition for about six months, and inquires whether he can be cured. This question, gentlemen, cannot be answered by a simple affirmative or negative. The prognosis of alopecia areata depends upon a number of factors, the most important of which is the age of the patient. The extent and duration of the baldness are of lesser importance. The younger the patient the more favorable the prognosis. Children are nearly always cured, adolescents and young adults frequently, but advanced adults rarely. Therefore the prognosis in this patient's case is unfavorable. Still, there is a chance of a cure resulting, and I would advise this man to place himself



**FIG. 6.—Alopecia areata.**



**FIG. 6.—Band form of alopecia.**





under treatment. We will prescribe for him to-day the following lotion:

R Tr. capsici,  
Tr. cantharidis, aa fʒss;  
Spts. vini recti, fʒii.—M.  
Sig.—To be brushed in twice a day.

Alopecia areata occasionally occurs in a "band form." This variety is typically represented in this woman (Fig. 6), who has been under observation for a number of months.

The alopecia extends in a broad band about three inches wide along the entire lateral aspect of the scalp. The disfigurement occasioned by this baldness is, as you see, very great. Unfortunately, this band form of alopecia is extremely refractory to treatment, and I fear very much that little or nothing can be done for this patient.

A word in conclusion as to the diagnosis of alopecia areata. In the presence of this condition I have often seen physicians suspect syphilis. There ought to be little difficulty in differentiating this form of alopecia areata from luetic alopecia. In the latter disease, the baldness does occasionally, though not commonly, occur in tolerably circumscribed areas; but these patches are totally unlike those of alopecia areata. In syphilis, the patches are irregular, "moth-eaten," not entirely devoid of hair, and the scalp is scaly, unhealthy looking, and studded with prominent follicles. Again, the hair of the entire scalp is usually implicated in the process.

In alopecia areata the patches are usually round or oval, perfectly bald, with smooth, soft skin, and follicles less prominent than normal. One or more patches may be present, but the unaffected portion of the scalp is perfectly healthy.



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